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INFECTIOUS NEURONITIS, PRESENT CONCEPTS OF ETIOLOGY AND TREATMENT

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Since 1916 when Guillain, Barré, and Strohl [1] first described a rapidly progressive motor and sensory polyradiculoneuritis with albuminocytological dissociation in the spinal fluid, numerous articles concerning this syndrome have appeared in the literature. It has been given various terms, including polyradiculoneuritis, myeloradiculitis, polyneuritis with facial diplegia, infectious polyneuritis, Guillain-Barré syndrome, and neuronitis. The last term was first mentioned by Mills in 1898 and reintroduced in 1919 by Foster Kennedy.[2] At that time, Kennedy reported four cases that presented unmistakable evidence of involvement of the spinal roots and the central nervous system, which he felt could be differentiated from other polyneuritides. We are inclined to agree with Kennedy and feel that the terms infectious neuronitis or Guillain-Barré syndrome are preferable in the designation of this condition.

The cause of neuronitis is not known. It has been attributed to a toxic or infectious agent, either bacterial or viral. Nutritional deficiency has been frequently mentioned as a cause, and, in recent years, some type of sensitivity or allergy has been suspect. All attempts to identify an infectious agent have been unsuccessful, though recently Bergamasso and Bottiglioni stated that they have cultural evidence suggesting that the Guillain-Barré syndrome is due to a neurotropic virus. The introduction of dimercaprol (2,3-dimercaptopropanol [BAL]) in the treatment of arsenical intoxication and the demonstration that its effect was probably due to the fact that it restored enzymatic equilibrium led to the concept that various neuropathies might be due to a disturbance of enzyme metabolism of the neurons. Peters and his associates showed that lewisite poisoned the pyruvate-oxidase system and that its action was due to its avidity for the sulfhydril groups of proteins in the enzymes that were essential to biological oxidation and reduction. It should be pointed out that many other enzyme systems occur in which the coenzymes contain members of the vitamin B complex and that the specific proteins to which these are attached may also contain the sulfhydril group. Thus inhibition of the pyruvate-oxydase system can be caused either by interference with the protein component of the system or by a disturbance of a coenzyme essential for pyruvate oxidation.

Joiner and his co-workers [3] reported on 16 hospital patients with polyneuritis and/or myelopathy who showed abnormal rise in blood pyruvate levels after glucose administration, indicating inhibition of this enzyme system. Thirteen of these patients with impaired pyruvate tolerance were given 100 mg. of thiamine daily for two weeks; six showed improvement in pyruvate metabolism. In these six patients, impaired pyruvate metabolism was attributed to defective intake or absorption of thiamine. A seventh patient showed clinical improvement without definite change in pyruvate metabolism after thiamine therapy. A course of dimercaprol was given to the remaining six who had failed to show any response either biochemically or clinically and to one of the original 16 who had been given thiamine before he was hospitalized. In two of these patients, clinical improvement coincided with return of the pyruvate level to normal; in a third, clinical and biochemical improvement followed the administration of dimercaprol but was not attributed to it. The other four showed no changes. On the basis of this evidence and the fact that

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1. Guillain, G.; Barré, J. A., and Strohl, A.: Sur un syndrome de radiculonévrite avec hyperalbuminose du liquide céphalo-rachidien sans réaction cellulaire: Remarques sur les caractères cliniques et graphiques des réflexes tendineux, Bull. et mém. Soc. méd. d. hôp. de Paris **40**: 1462, 1916.
2. Kennedy, F.: Infective Neuronitis, Arch. Neurol. & Psychiat. **2**: 621 (Dec.) 1919.
3. Joiner, C. L.; McArdle, B., and Thompson, R. H. S.: Blood Pyruvate Estimations in the Diagnosis and Treatment of Polyneuritis, Brain **73**: 431 (Dec.) 1950.

dimercaprol has been reported as benefiting various polyneuritides,[4] it seems reasonable to suggest that neuronitis as well as polyneuritis may be due to a disturbance of enzyme systems of the neurons. It is suggested that this enzyme disturbance may be due to a toxic or allergic reaction, and, therefore, that neuronitis is not a specific disease but a symptom-complex that may have various causes. This idea is supported by the fact that the Guillain-Barré syndrome has been reported in connection with infectious mononucleosis, diphtheria, porphyria, after various infections, and after vaccination for smallpox and Japanese B encephalitis, and with tetanus toxoid.

The condition generally develops a few days to weeks after a mild infection. There first appear paresthesias and pains in the extremities, usually the lower ones, with rapid appearance of motor weakness that may be more marked proximally and may first be manifest in the upper extremities. Facial paralysis, either unilateral or bilateral, may appear at any time. Occasionally, this is the first sign of motor involvement. Spinal cord symptoms, including sphincter disturbances, are found especially in severe cases. Sensory changes may be present but are not marked. Tendon reflexes are diminished or absent, and there may be pathological toe signs. There is nerve trunk tenderness. Fever is usually not present. Spinal fluid examination shows clear fluid (rarely xanthochromic) under normal pressure. Typically, there is definite elevation in total protein without a proportionate increase in cells. Recovery is the rule, unless respiratory paralysis develops. Guillain in 1936 maintained that all patients should recover. In 1937, according to Hand and Rudoy, he (Guillain) "revoked his opinion of a year before without reservation." That the prognosis is often grave is now generally accepted. Forster, Brown, and Merritt [5] report a mortality rate of 42% in 26 cases; Roseman and Aring [6] report 18.8% in 16 cases; and Gilpin, Moersch, and Kernohan report 14% in 35 cases.

Pathological changes are most marked in the peripheral and cranial nerves. The primary and most characteristic feature, according to Scheinker,[7] is the pronounced swelling of the nerve fibers in the spinal roots and spinal cord tracts and swelling of the cranial and peripheral nerves. The sudden increase in bulk of most of the spinal roots, caused by a tremendous swelling of the axons, leads to narrowing and finally to obliteration of the perineural spaces. The mechanical constriction of the strangulated radicular trunks may result in sudden loss of function. The plugging of the perineural spaces blocks absorption of spinal fluid along these channels and results in the trapping and stagnation of the spinal fluid, with a consequent increase in total protein.

Forster, Brown, and Merritt [5] state that the chief pathological change is degeneration of the peripheral nerves, with slight changes in the axis cylinders. Inflammatory reactions in the nerves may or may not be present. The central nervous system is not affected to any appreciable extent, except for swelling and chromatolysis (axonal reaction) of the motor cells in the medulla and ventral horns of the spinal cord. Haymaker and Kernohan [8] are in accord with this. Lowenberg and Foster,[9] however, feel that the condition is characterized by "primary degeneration of the myelin in the peripheral nerves, spinal ganglia, cord, and brain stem." They state, "the equally severe involvement of efferent and afferent pathways and the disregard for synapses exclude the possibility of secondary degeneration within the central nervous system arising from primary disease of the peripheral nerves."

Treatment, influenced by changing concepts as to etiology, has varied considerably. Since the condition tends to be self-limiting, it is difficult to evaluate the effects of any type of therapy. In recent years, treatment has been largely palliative; special attention has been devoted to the prevention of respiratory and other complications. Straus and Rabiner and more recently Rabiner, Rosenberg, and Freedman have recommended typhoid vaccine therapy. Various elements of the vitamin B complex have generally been administered. Nielsen,[4a] Creaturo,[4b] and Furmanski have reported favorable results from the use of dimercaprol. Stillman and Ganong report one case in which corticotropin (ACTH) at first seemed to be effective for 15 days. The patient then relapsed, was given cortisone, and recovered. They used these preparations on the theory expressed by Thorn that adrenocorticotrophic hormones interfere with allergic reactions, though Thorn also states that an excess of the same drugs plays a role in the inactivation of the sulfhydril groups. On the basis of our experience, we feel that the best results are obtained by the use of vitamin B and dimercaprol and, in those cases in which respiratory and swallowing difficulty develops, by the prompt use of the respirator, usually in conjunction with tracheotomy.

Twenty-three patients, who have been observed in the past three years, are reported in this paper. Eight required respirator care. There were two deaths, both in patients who had respiratory difficulty. In one instance, no respirator was available; in the other, death resulted from technical difficulties and might otherwise have been avoided. The results obtained in these patients do not differ materially from those reported in the literature, except that the mortality has been markedly reduced in those requiring respirator care. The salient features in each of the 23 cases follow.

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5. Forster, F. M.; Brown, M., and Merritt, H. H.: Polyneuritis with Facial Diplegia: Clinical Study, New England J. Med. **225**: 51 (July 10) 1941.

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REPORT OF CASES

CASE 1.—Numbness and tingling developed in the hands and feet of a 34-year-old cement worker. There were no antecedent respiratory or gastrointestinal symptoms. Paresthesias had extended to his legs and arms by the fourth day, and weakness of the extremities was noted. By the sixth day, he could not walk, and facial weakness appeared. There were no sphincter disturbances. Examination revealed an alert, cooperative patient in no acute distress. His temperature was normal. He was unable to either close his eyelid or retract his mouth on the right, but there were no other cranial nerve abnormalities. Mild nuchal rigidity and paravertebral muscle spasm were present. There was poor respiratory control, and force of cough was diminished. Respiratory exchanges seemed adequate. There was severe weakness of all extremities that was marked more proximally in the upper extremities and more distally in the lower. Tendon reflexes were hypoactive. No superficial reflexes or pathological toe signs were elicited. Sensation was intact. The spinal fluid was clear and colorless; the initial pressure 240 mm. of water; lymphocytes 6 per cubic millimeter; and total protein 257 mg. per 100 cc.

On the 11th and 12th days, 250 mg. of dimercaprol was administered every six hours, but weakness increased until the patient was unable to turn in bed or to lift his hands from his sides. On the 16th day, dimercaprol therapy was reinstituted and continued (250 mg. every 12 hours) for one week. Improvement was gradual, but by the ninth week the patient could walk with assistance. Twelve weeks after onset of the illness, he returned to his regular work. When last contacted, two years later, there were no residual effects of his illness.

CASE 2.—This 41-year-old waiter had no initial respiratory or gastrointestinal symptoms. While at work, numbness and weakness of his left face developed, and he could not close his left eye. A few hours later, his legs and arms felt weak, and he experienced numbness and "shooting" pains in his knees, thighs, and shoulders. The next day, weakness developed on the right side of the face and the generalized weakness increased. The shooting pains persisted, and numbness and tingling of the feet and hands developed. After one week, he was unable to walk. About one month later, it was noticed that the muscles in his arms and legs, especially his thighs, were twitching.

Examination showed bilateral facial weakness and paresis of all extremities, which was more marked proximally. Tendon and superficial reflexes were absent. Response to plantar stimulation was normal. There was nerve trunk tenderness but no significant sensory deficit. Sphincter control was normal. Spinal fluid was clear and xanthochromic, with normal dynamics, 2 lymphocytes per cubic millimeter, total protein 179 mg. per 100 cc., and negative serologic tests. An electromyogram revealed diffuse and severe lower motor neuron damage in all extremities and in the face.

Pain was so severe that narcotics were required. By the end of four months, muscular atrophy and fasciculation in the extremities were marked. At this time, the patient was given a 10 day course of dimercaprol in conjunction with large amounts of vitamins. No significant objective improvement resulted, although the patient felt considerably better. He later became ambulatory but required canes. After 18 months, mild weakness of the face and extremities persisted, but he was fully ambulatory and able to work.

CASE 3.—The illness of this 59-year-old housewife began with anorexia, malaise, sore throat, fever, and chills, for which she was given penicillin, aureomycin, and chloramphenicol. Four weeks later, as she was recovering from these symptoms, weakness appeared on the right side of the face. Three days later, mild weakness of the extremities was noted, which was most apparent in walking. She was unable to urinate. Physical examination revealed several abnormalities. The retinal veins were engorged, and the optic disk margins were blurred. There was bilateral facial weakness, which was severer on the right. Minimal nuchal rigidity was present. Weakness of the extremities was slight. Biceps, triceps, and ankle jerks were diminished; knee jerks were active. Superficial reflexes were absent. Babinski and Chaddock signs were elicited bilaterally. There was no sensory deficit, but there was mild aching of the calf and thigh muscles. Spinal fluid was clear and colorless, with normal dynamics, 6 lymphocytes per cubic millimeter, total protein 100 mg. per 100 cc., and a negative Wassermann reaction. An electromyographic study was not done. After two weeks with no specific treatment, the patient improved rapidly and completely recovered after three months.

CASE 4.—Constipation and "nervousness" were chronic problems to this 25-year-old woman. After a two day period of nausea, vomiting, feverishness, and malaise, she noticed extreme weakness of her legs that was associated with numbness and aching. By the end of the week, she could no longer walk unassisted, her upper extremities became weak, and the aching muscular pain in her legs and back became severe. Examination on the 11th day of illness revealed an alert, well-oriented, cooperative, but apprehensive young woman in acute distress.

There was an inconstant diplopia on left lateral gaze. The other cranial nerves were intact. Moderate nuchal rigidity and paravertebral spasm were present. Weakness was generalized, but was more marked in the lower extremities. Tendon reflexes were hypoactive. Superficial reflexes were absent. Babinski's sign was elicited bilaterally. There were nerve trunk and muscle tenderness in the lower extremities, slight hypesthesia distally, and hyperesthesia over the upper thighs and buttocks. There was some difficulty in urination. The spinal fluid was clear and colorless, with normal dynamics, 1 lymphocyte per cubic millimeter, total protein 270 mg. per 100 cc., and a negative Wassermann reaction. An electromyogram demonstrated diffuse lower motor neuron damage in all extremities. Treatment consisted of vitamins, sedation for anxiety and restlessness, codeine for leg and back pains, and psychotherapy. At the end of two months, the patient was fully ambulatory and was discharged from the hospital much improved.

CASE 5.—One month after symptoms of coryza, sore throat, and brassy cough, this 57-year-old man noted a burning sensation over his knees and thighs. Three days later his legs felt weak and ached on exertion. By the 10th day, numbness, which had begun in the toes, spread upward to the level of the umbilicus. The weakness became more marked, and the patient could no longer urinate. The cranial nerves were intact. There were no meningeal signs. His strength appeared normal in the upper extremities. Muscles of the lower extremities showed moderate weakness. Tendon reflexes were normal in both of the upper extremities and in the left lower extremity but hyperactive in the right knee and ankle. Superficial reflexes were absent. Babinski's sign was elicited bilaterally. There was a zone of hyperesthesia from the 10th to 12th dorsal segment. Above this zone, sensation was normal, and below it pain, touch, and vibratory perception were diminished. Catheterization was required because of urinary retention. Spinal fluid was clear and xanthrochromic, with normal dynamics, 3 lymphocytes per cubic millimeter, total protein 51 mg. per 100 cc., and a negative Wassermann reaction. An electromyogram demonstrated diffuse lower motor neuron damage in all extremities.

The patient was given 500 mg. of aureomycin every six hours for two weeks and large amounts of thiamine, niacin, and brewers' yeast. After 24 hours, he was able to void and urinary residuals were small. In the next few days, improvement was marked. Four weeks after onset of the condition, he had only mild weakness and numbness. After three months, slight residual weakness and numbness of toes persisted.

CASE 6.—Three weeks after the development of upper respiratory symptoms including malaise, anorexia, and sore throat, this 74-year-old woman noted difficulty in walking, numbness of her feet, and urinary retention. Examination revealed an alert, well-oriented, cooperative, but anxious and suggestible elderly woman. Her blood pressure was 210 systolic and 110 diastolic, and her pulse was 92. Her heart was slightly enlarged and showed an apical systolic murmur. Her tongue was red and "avitaminotic." No cranial nerve abnormalities or meningeal signs were found. Initially, the generalized weakness was thought to be hysterical in origin, because there was a marked variation in performance and because a full range of movement was present. Tendon reflexes were absent. Ab-

dominal reflexes were hypoactive. Babinski's sign was positive bilaterally. There was nerve trunk tenderness. Vibration sense was absent below the knees. Position sense was absent in the toes. Urinary retention necessitated catheterization. Spinal fluid was clear and colorless, with normal dynamics, no lymphocytes, total protein 139 mg. per 100 cc., and a negative Wassermann reaction. An electromyogram demonstrated diffuse lower motor neuron damage in all extremities. Two weeks after onset of weakness, gradual improvement was observed. At the end of three months, the patient was able to walk with assistance.

Case 7.—One week after a short episode of malaise, headache, mild diarrhea, and feverishness, this 40-year-old man had difficulty in rising, standing, and walking. His hands and feet became numb, and weakness rapidly increased until he could not get out of bed. He was cared for at home, but, after three weeks, nuchal rigidity was noted, and he was admitted to the hospital. Examination revealed an alert, cooperative man in no distress. The cranial nerves showed no abnormalities. There was slight nuchal rigidity, moderate hamstring, and paravertebral muscle spasm. The muscles of all the extremities and of the neck were very weak. Tendon and superficial reflexes were normal. No unusual plantar responses were elicited. There were no sphincter disturbances. Spinal fluid was clear and colorless, with normal dynamics, 5 lymphocytes per cubic millimeter, total protein 145 mg. per 100 cc., and a negative Wassermann reaction. Treatment consisted of supportive vitamin therapy and continued bed rest. After two days, improvement began. By the end of three weeks the patient was fully recovered functionally and returned to work six weeks after onset of weakness.

Case 8.—Four days after the beginning of nasal congestion, coryza, and sore throat, this 57-year-old man experienced difficulty in standing and walking and had aching in his thigh muscles. The weakness of his lower extremities became severe, and he required assistance in walking. Because of a concomitant personality disorder, a diagnosis of hysteria was made. Ten days after weakness was noticed, he was unable to walk, had difficulty urinating, and was constipated. He was alert, well oriented, and cooperative. His blood pressure was 150 systolic and 90 diastolic. No cranial nerve abnormalities or meningeal signs were found. Strength in the upper extremities was good, but there was moderately severe weakness of all muscle groups in the lower extremities. Tendon reflexes were normal in the upper extremities and absent in the lower extremities. Superficial reflexes were intact. No abnormal plantar responses were elicited. There was nerve trunk tenderness in the legs. He experienced difficulty in urinating, but urinary residual was not determined. Spinal fluid was clear and colorless, with normal dynamics, 12 lymphocytes per cubic millimeter, and total protein 129 mg. per 100 cc. A spinal Wassermann reaction was not reported; blood Wassermann was positive. Roentgenograms of the pelvis, lumbosacral spine, and chest revealed nothing abnormal. An electromyogram demonstrated diffuse lower motor neuron damage in the lower but not in the upper extremities. Without special therapy, the patient's strength improved, his knee jerks returned, and he became ambulatory. He subsequently received 10 million units of penicillin for syphilis. Prostatic studies did not demonstrate carcinoma. By the end of three months, recovery was complete.

Case 9.—Numbness and tingling developed in the fingers of a 65-year-old man with poorly controlled diabetes. Four days later, these paresthesias developed in his feet, and he noted weakness in his legs. After 10 days, there was marked muscle weakness in all extremities. There were no sphincter disturbances. Physical examination revealed a blood pressure of 160 systolic and 80 diastolic but otherwise normal. Neurological examination showed no cranial nerve abnormalities and no meningeal signs. The right grip was slightly stronger than the left. No deep or superficial reflexes were elicited. Plantar responses were normal. Two-point discrimination was impaired in the fingers. Touch perception was diminished in the feet and distal third of the legs. Nerve trunk tenderness was present in the legs. Sphincters were intact. Spinal fluid was clear and colorless, with normal dynamics, 2 lymphocytes per cubic millimeter, total protein 184 mg. per 100 cc., and a negative Wassermann reaction. Treatment was begun on the 10th day of the patient's illness and included administration of thiamine, vitamin B_{12}, and crude liver extract. Dimercaprol was given in amounts of 150 mg. every six hours for seven days. His course continued downhill until, after 10 days more, he was severely paralyzed, then his strength gradually improved. After six months, bilateral foot drop and weakness of the legs and hands persisted. His recovery was incomplete.

Case 10.—Two days after the extraction of three carious teeth, a 46-year-old woman noticed numbness and tingling of her hands that was followed shortly by marked bilateral weakness of grip and numbness of her lower extremities. Shortly thereafter, her legs became weak, and, within a few hours, she could no longer walk. Her hips and shoulders became painful. Examination revealed an alert, well-oriented, cooperative woman who was suggestible and very tense but in no acute distress. There was slight enlargement of her thyroid gland. No cranial nerve abnormalities and no meningeal signs were detected. At first, objective weakness was minimal, but after 12 days it became severe. By this time, bilateral facial weakness and respiratory difficulty had developed. The tendon reflexes were hypoactive in the upper and inconstant in the lower extremities. Superficial reflexes were absent. No abnormal plantar responses were elicited. Sensation was intact, except for nerve trunk and muscle tenderness. There was no sphincter disturbance. Spinal fluid was clear and colorless, with normal dynamics, 3 lymphocytes per cubic millimeter, total protein 360 mg. per 100 cc., and a negative Wassermann reaction.

Beginning on the 12th day, dimercaprol was given in amounts of 200 mg. three times daily for three days and then daily for two weeks. Vitamins and crude liver extract were also administered. The patient gradually recovered her strength until, at the end of two months, she could walk with assistance, had a good grip with both hands, and there was no evidence of facial weakness. Paresthesias of the feet persisted, and, 14 months after onset, there was still slight residual weakness.

Case 11.—In a 27-year-old alcoholic man, initial symptoms of nausea, vomiting, malaise, muscular aching, and low back pain were followed in three days by generalized weakness, which was most noticeable in the lower extremities. In seven days, this weakness progressed until he could not walk or get out of bed. There were no paresthesias or other difficulties. Examination revealed an alert, cooperative, tense, poorly nourished man with no significant physical abnormalities. There were no cranial nerve abnormalities or meningeal signs. Weakness was present in all extremities but was severest in the thigh muscles. Tendon reflexes were normal in the upper and hypoactive in the lower extremities. No abnormal toe signs were elicited. Calf tenderness was bilateral. No sensory deficits were detected. Spinal fluid was clear and colorless, with normal dynamics, 23 lymphocytes and 1 neutrophil per cubic millimeter, total protein 200 mg. per 100 cc., and a negative Wassermann reaction. An electromyogram demonstrated diffuse lower motor neuron damage in all extremities.

After four days of intensive vitamin therapy, the weakness continued to increase. Dimercaprol was administered in doses of 200 mg. every six hours for two weeks. At the end of the second week, definite improvement was observed, and the patient gradually became ambulatory. After five weeks, he was discharged from the hospital with mild, persistent weakness and unsteadiness of gait, but he was markedly improved.

Case 12.—Numbness and tingling of hands and feet developed insidiously in a 53-year-old man. These developments were followed shortly by an unsteady gait and gradually by progressive weakness of the lower extremities. By the end of three weeks, he was unable to walk unassisted and his grip was weak bilaterally. Examination revealed an alert, suggestible, moderately uncooperative, malnourished man in no distress. His general physical condition was not remarkable. His pupils were small and unequal, the left pupil was larger than right, and they both reacted slowly to light and to convergence test. His face was mask-like, and his facial movements were weak.

Gag reflex was hypoactive. There were no other cranial nerve abnormalities and no meningeal signs. All movements of the extremities were very weak. Tendon reflexes were absent throughout. Superficial reflexes were normal. There were no abnormal plantar responses. Hyperesthesia of feet and nerve trunk and muscle tenderness were marked. There was slight hypalgesia of the hands and feet. Sphincters were normal. Spinal fluid was clear and colorless, with normal dynamics, 1 lymphocyte per cubic millimeter, total protein 87 mg. per 100 cc., and a negative Wassermann reaction. An electromyogram demonstrated moderately severe lower motor neuron damage in all extremities. After four days of intensive vitamin therapy, 200 mg. of dimercaprol every six hours was given for one week. By the end of this period, the patient began to ambulate with assistance; subsequent improvement was gradual. When seen five months after onset, he was able to walk fairly well but showed residual weakness of the thighs and arms.

CASE 13.—The patient was a 39-year-old woman. Six weeks after a mild upper respiratory illness, numbness developed in her fingertips. Over a 24 hour period, the numbness spread upward to elbow, neck, face, and tongue. Slight weakness was noted in the upper extremities. The next day, similar paresthesias and weakness began in her toes and ascended to her thighs and lower trunk. By the 12th day, her grip was very weak, and her legs would not support her weight. Also, difficulty in swallowing was noted for the first time. Since she did not appear in great distress, her husband felt that she was malingering. His opinion led to marked emotional disturbances in the patient. Examination showed an alert, cooperative, apprehensive, tense woman in no acute distress. She could not close her eyes or grimace. The sternomastoid muscles were weak bilaterally. The remaining cranial nerves were intact, and no meningeal signs were elicited. There was severe weakness of all extremities. Superficial reflexes were absent. Plantar responses were normal. Mild hypalgesia of the hands and feet and moderate tenderness of the leg and thigh muscles were found. Spinal fluid was clear and colorless, with normal dynamics, 28 lymphocytes per cubic millimeter, total protein 300 mg. per 100 cc., and a negative Wassermann reaction. An electromyogram demonstrated diffuse lower motor neuron damage in all extremities. Despite intensive vitamin therapy, by the end of five weeks there had been no appreciable improvement. Dimercaprol was administered in doses of 100 mg. daily for two weeks. After one week, definite improvement was noted, and, at the end of two weeks, the patient was able to walk without assistance. Three months after onset of numbness, she was well.

CASE 14.—While at a football game, this 29-year-old woman became chilled, and nasal congestion, sneezing, coryza, and general malaise, but no fever, developed. Two days later, she awoke with marked weakness of both legs. By the fourth day, she was unable to walk and had a mild suboccipital headache. Weakness of her arms and lips and slight difficulty in swallowing was noted. Examination revealed a listless, withdrawn, disinterested, but fairly cooperative woman lying quietly in bed in no distress. There was bilateral facial weakness and moderate dysphagia. Slight nuchal rigidity and paravertebral and hamstring spasm were noted. There was severe weakness of all extremities, which was more marked distally. No tendon or superficial reflexes and no plantar responses were elicited. There was pronounced nerve trunk and muscle tenderness but no sensory deficits. Evidence of diaphragmatic weakness appeared, and vital capacity was reduced to 1,450 cc. Sphincters were intact. Spinal fluid was clear and colorless, with normal dynamics, no lymphocytes, total protein 130 mg. per 100 cc., and a negative Wassermann reaction. An electromyogram demonstrated diffuse lower motor neuron damage in all extremities. One week after onset of the illness, 120 mg. of dimercaprol was given every six hours for three days; then the same amount was given every 12 hours for 10 days. The first evidence of improvement was noted one week later. Six weeks after onset, the patient could walk with assistance and could close her eyelids completely. After eight weeks, she was nearly completely recovered.

CASE 15.—Eight days after symptoms of a "head cold," severe nausea and vomiting suddenly developed in a 57-year-old bartender and persisted for 24 hours. He then noticed a numbness of his feet that spread through all his extremities and became associated with generalized weakness. Eleven days after onset of the paresthesias, difficulty in swallowing and pain across the lower anterior chest, which was associated with effort breathing, developed. Bilateral facial weakness appeared and was followed by slurred speech, diplopia, and severe respiratory embarrassment. Examination revealed an apprehensive, restless, moderately cyanotic, extremely weak, but cooperative white man with rapid, shallow respirations. His rectal temperature was 100 F, pulse 104, and respiration 32. His vital capacity was 600 cc. Weakness of the extraocular muscles (unspecified), weakness of the masseters bilaterally, and inability to wrinkle the forehead, close the eyelids, and purse the lips were observed. He was unable to swallow or to clear away throat secretions and could only whisper weakly. He was unable to raise his head from the pillow. The neck muscles were flaccid, but there was hamstring spasm. Movements of the arms and hands were very weak. There was only slight ankle and toe movement. There were no tendon or superficial reflexes and no plantar responses. Sensation was intact. Urinary retention was present. Spinal fluid was clear and colorless, with normal dynamics, 2 lymphocytes per cubic millimeter, total protein 320 mg. per 100 cc., and a negative Wassermann reaction. A tracheotomy was performed, and the patient was placed in a respirator with both positive and negative pressure. Initially, 450 mg. of dimercaprol was administered and followed by 300 mg. of the drug every four hours for two days and then 225 mg. every six hours for four days. Despite a stormy course, the patient was able to swallow and to close his eyes weakly after one week in the respirator. His vital capacity, facial movements, swallowing, speech, and the strength of his extremities gradually improved. After six weeks, he no longer required a respirator, and, after nine weeks, he was able to walk with assistance. Three months after onset, he could walk unassisted but still had considerable weakness of the extremities.

CASE 16.—The initial symptoms in this 54-year-old woman were sore throat, coryza, malaise, nausea, vomiting, and mild diarrhea. These symptoms subsided after four days, but, after eight days, her temperature rose to 100.4 F and diplopia, ptosis, and weakness of lower extremities developed. By the 12th day, she had difficulty swallowing, breathing, and moving her upper extremities, in addition to numbness of her hands and pain in her calves. Examination at that time showed a confused, apprehensive, somewhat irrational, dyspneic, mildly cyanotic woman who appeared critically ill. Her temperature was 97 F, pulse 96, and respirations 30. Bilateral ophthalmoplegia with ptosis, weak jaw movements, bilateral facial and palatal weakness, dysphagia with pooling of pharyngeal secretions, poor gag reflex, and weak phonation were noted. There was poor diaphragmatic power, and the intercostal and accessory muscles were weak. The muscles of all the extremities were very weak, although motion was possible in the toes, ankles, fingers, and wrists. Tendon reflexes were hypoactive throughout. Superficial reflexes were absent. Babinski's sign was elicited on the left side. Calf tenderness was marked, but there were no sensory deficits. Sphincters were intact. Spinal fluid was clear and colorless, with normal dynamics, 7 lymphocytes and 1 polymorphonuclear leukocyte per cubic millimeter, total protein 94 mg. per 100 cc., and a negative Wassermann reaction. An electromyogram showed diffuse lower motor neuron damage.

Treatment included tracheotomy, use of a respirator with continuous oxygen and suction, Levin tube feedings, and administration of large doses of vitamins, and 250 mg. of dimercaprol every six hours for 10 days. After 10 days, the respirator was no longer required continuously, and oral feedings were possible. After two weeks, use of the respirator and tracheotomy was discontinued. After three weeks in the hospital, the patient was ambulatory with assistance and was discharged. Two months after onset of the illness, she walked well without assistance. The only residual effects were mild persistent diplopia and a slight ptosis of the right eyelid.

CASE 17.—Two months after a "head cold" and a severe exacerbation of asthma, numbness and weakness of legs and hands developed in this 55-year-old male alcoholic. Over a 12 hour period, the numbness and weakness progressed until he could not arise or turn in bed. He had aching pains in his legs and arms and was unable to void. Ten days later increasing respiratory distress and difficulty in swallowing necessitated hospitalization. Examination showed an alert, cooperative man with rapid, shallow respirations who was in moderate distress. His right pupil was small but reacted to light. There was slight limitation of upward and right lateral conjugate eye movements, as well as bilateral facial weakness and slight dysphagia. Diaphragmatic, intercostal, and accessory respiratory movements were very weak. Vital capacity was 550 cc. and amount of tidal air 350 cc. The lower extremities were severely paretic, more so on the right than on the left side. There was only slight weakness in the upper extremities. Tendon reflexes were absent except for normal biceps action bilaterally. Abdominal and cremasteric reflexes were present. No plantar signs were elicited. An indwelling catheter was required. Spinal fluid was clear and colorless, with normal dynamics, 4 lymphocytes per cubic millimeter, total protein 200 mg. per 100 cc., and a negative Wassermann reaction. A roentgenogram of the chest showed an infiltrate at the right lung base consistent with pneumonitis. Treatment included management in a respirator and administration of 100 mg. of dimercaprol twice daily for two weeks, vitamins, and fluids intravenously. After three weeks in the respirator, the patient was able to remain out of it for several hours without difficulty. After five weeks, use of the respirator was entirely discontinued. The patient was discharged from the hospital shortly thereafter and, after 10 weeks, residual effects were minimal.

CASE 18.—Three weeks prior to the onset of weakness, this 31-year-old man had an upper respiratory infection from which he recovered after a few days. The weakness was associated with numbness, tingling, and "deadness" of fingers and toes. By the third day, he was unable to get out of bed and, subsequently, could not feed himself or turn in bed. By the eighth day, progressive breathing and swallowing difficulty made hospitalization necessary. Examination at that time showed an apprehensive, poorly cooperative man with marked respiratory embarrassment, who appeared critically ill. His temperature was 102 F (rectal), pulse 120, and respirations 28. There was bilateral facial paralysis. He was unable to swallow or clear his pharynx of secretions. He spoke in a coarse whisper and could not lift his head from the pillow. No diaphragmatic movements could be demonstrated and, although the other respiratory muscles were weak, they were active. His vital capacity was 600 cc. and the amount of tidal air 200 cc. All the extremities were paralyzed except for slight movement of the fingers. No tendon reflexes or toe signs could be elicited. There was some vague impairment of sensory reaction to pinprick in the lower extremities. There was urinary retention. Spinal fluid was clear and colorless, with normal dynamics, 5 lymphocytes per cubic millimeter, total protein 94 mg. per 100 cc., and a negative Wassermann reaction.

Treatment included tracheotomy, use of a respirator with positive as well as negative pressure, intravenous and Levin tube feedings, and administration of 150 mg. of dimercaprol every six hours for five days. The patient was intermittently delirious and febrile for two weeks. After that, his condition improved and he achieved a complete recovery.

CASE 19.—A 27-year-old woman had a severe systemic illness, which was diagnosed as infectious mononucleosis and which was followed by malaise and drowsiness. Five months later facial weakness developed on the right side and was followed in five days by facial weakness on the left. Two weeks later, weakness of the right arm developed, and the patient was sleepy most of the time. She could not raise her head from the pillow, had difficulty swallowing, and regurgitated fluid through her nose. She complained of an unpleasant drawing sensation on her right side. Examination at that time revealed a mild, divergent strabismus when she looked to the right. There was a complete bilateral facial palsy. The soft palate rose poorly on phonation and the gag reflex was absent. Her neck muscles were very weak. There were no meningeal signs. There was severe weakness of the right arm, although finger movements were possible. Tendon reflexes were present but hypoactive in the right upper extremity, and abdominal reflexes were present. Spinal fluid at this time was normal. Two weeks later, an electromyogram showed complex motor units in all lumbosacral segments and fibrillation of denervation in cervical segments. After six weeks, both right extremities were very weak, and, after seven weeks, all extremities were quite weak, and respiratory difficulty developed rapidly. Examination revealed elevation and paralysis of the right diaphragm and bilateral abductor paralysis of larynx. A tracheotomy was performed, and the patient was placed in a respirator. She was severely ill and mildly euphoric. The respirator was required for three months. After five and a half months, she was ambulatory and was discharged with mild residual weakness of the right lower extremity and some generalized weakness. Her recovery was incomplete.

CASE 20.—Nausea, vomiting, anorexia, and malaise in a 29-year-old woman were followed, after three days, by paresthesias and severe weakness of all extremities. Bilateral facial weakness appeared at the end of one week. After two weeks, there was rapid onset of dysphagia and severe respiratory weakness. Other than bilateral facial weakness, there were no cranial nerve abnormalities. There were no meningeal signs. There was profound weakness of all extremities, but the sphincters were intact. All tendon reflexes were absent, but abdominal reflexes and plantar responses were normal. Spinal fluid was clear and colorless, with normal dynamics, 2 lymphocytes per cubic millimeter, total protein 209 mg. per 100 cc., and a negative Wassermann reaction. Treatment included tracheotomy, use of a respirator, and administration of 150 mg. of dimercaprol twice daily for two days and then 100 mg. twice daily for two weeks. The tracheotomy and respirator were required for six weeks, after which the patient's improvement was rapid. Her recovery was complete after three months.

CASE 21.—Four weeks before appearance of weakness, abdominal distress, nausea, and vomiting developed in this 68-year-old woman. She recovered from this, but, 10 days before onset of weakness, shortness of breath, a productive cough, and pleural effusion developed. Severe numbness and prickling sensations over hands and feet were followed by quadriplegia that gradually progressed over a four week period. During the fifth week, there was a rapid development of a severe respiratory weakness and urinary incontinence. Examination at that time revealed no cranial nerve abnormalities or meningeal signs. Only slight movement of fingers and toes was possible. No tendon or superficial reflexes and no plantar responses could be obtained. There was mild hypalgesia, distal to wrist and knees, and marked calf tenderness. Spinal fluid was clear and colorless, with normal dynamics, 3 lymphocytes per cubic millimeter, total protein 113 mg. per 100 cc., and a negative Wassermann reaction. During the first month of her illness, this patient received daily an average of 30 μg of vitamin B_{12}. At the end of this time, 200 mg. of dimercaprol was given every six hours for 10 days. Despite the use of this drug, symptoms progressed, and, at the end of five weeks, a respirator was required. The patient was severely ill and was delirious part of the time. Return of respiratory function was gradual, and the patient required the respirator for five weeks. Her recovery was then rapid, and she was able to walk with assistance three months after onset. After five months, the only residual effects were mild weakness and numbness of the lower extremities.

CASE 22.—The first symptoms of illness in this 57-year-old woman were tingling and weakness of her upper and later her lower extremities. These symptoms progressed gradually over a three month period. The progression was more rapid during the last two weeks of this three month period, and difficulty in speaking, swallowing, and breathing developed the day before the patient was admitted to the hospital. There was facial weakness on the right side, dysphagia, dysphonia, and weakness of the neck muscles. The tongue deviated to the left

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on protrusion. There were no meningeal signs. Weakness was marked in all extremities but was greater on the right side. No tendon reflexes or pathological toe signs were elicited. Calf tenderness, but no other sensory abnormality, was demonstrated. Spinal fluid was clear and colorless, with normal dynamics, no cells, total protein 200 mg. per 100 cc., and a negative Wassermann reaction. An electromyogram demonstrated diffuse lower motor neuron damage in all extremities. Levin tube feedings were required, and vitamins and crude liver were administered. Ten days after the patient was admitted, her temperature was 101 F, and there were moist rales in both lungs. On the 12th day, she vomited and aspirated some of the vomitus; severe cyanosis ensued. Respirations were inadequate subsequently, but a respirator was not available. The patient died of severe pneumonia three days later.

CASE 23.—For two months prior to onset of weakness, this 34-year-old man had been drinking excessively and had had a chronic cough and intermittent "head cold." He first noted unsteadiness of gait, numbness, and tingling of his feet. During the following week, there developed severe quadriparesis, mild facial weakness on the left side, difficulty in swallowing, speaking, and breathing, and paresthesias of hands and feet. Examination revealed an alert, cooperative, somewhat apprehensive man in no distress. His temperature was 100 F (rectal), pulse 120, and respirations 24. His blood pressure was 155 systolic and 110 diastolic. The left facial muscles were weak. His voice was muffled and had a nasal quality. There was dysphagia with inability to clear secretions from pharynx. He could not lift his head from the pillow, and his tongue deviated slightly to the left. Respiratory movements were shallow and weak. The muscles of all extremities were very weak but more so in the lower extremities. No tendon reflexes or plantar responses were elicited. Urinary retention was present. There was hypalgesia over the lower part of the abdomen. Spinal fluid was clear and colorless, with normal dynamics, no cells, total protein 245 mg. per 100 cc., and a negative Wassermann reaction. A tracheotomy was performed, a Levin tube was inserted for feeding, and the patient was placed in a respirator. He was given large amounts of vitamins, as well as 200 mg. of dimercaprol every six hours. After several days during which weakness progressed, his condition stabilized, and slight improvement was noted. After aspiration of vomitus, however, he became cyanotic, profound shock developed and the patient died despite all measures. Autopsy revealed atelectasis of both lower lungs and large amounts of aspirated material.

COMMENT

From the data presented, it seems evident that these cases are representative of those described by Guillain, Barré, and Strohl. It should probably be pointed out that the majority of our patients had some type of antecedent infection or disturbance and in all but one symptoms developed within six weeks. Symptoms were largely combined motor and sensory, but the signs were predominantly motor. There was sphincter disturbance in nine patients, and this appeared to be the result of spinal cord involvement. There was facial paralysis in 14 cases (bilateral in 12) and spinal fluid protein was increased in all instances but one. In this one case, the spinal puncture had been performed early in the illness, and the patient refused another. In general, results of treatment were good. Twelve patients made a complete recovery, nine had residual effects (none of which were severe), and some are still showing improvement.

In one case (case 23), autopsy showed lymphocytic infiltration and some widening of the interstitial spaces in the peripheral nerves. No significant demyelination and no free fat were found. There was softening in the midthoracic region of the spinal cord, and sections through this area were severely frayed and showed marked loosening of the fibrous elements, with much interstitial edema. Only a few nerve cells could be identified. There was no definite demyelination, round cell infiltration, or free fat. Sections through the medulla and low pons, including a section through the seventh nerve, showed only moderate edema. The neuropathologist considered these changes to be consistent with the acute course of infectious neuronitis seen in this case.

With regard to treatment, it is difficult to assess the efficacy of vitamin B or dimercaprol. It would seem that dimercaprol may have a specific effect in some instances, since the patients in cases 10 to 14 seemed to show significant improvement after dimercaprol was administered. Also, all the seriously ill patients, except one who died, received dimercaprol. This could be considered as a factor in the high percentage of recovery obtained here.

There is some indication that avitaminosis may play a part in the development of neuronitis, since in four of our cases there was a history of excessive use of alcohol and in another there was a history of diabetes. The antecedent gastrointestinal disturbance with vomiting noted in six cases, combined here with upper respiratory infection in two and alcoholism in one, has been reported elsewhere [10] and may have induced some degree of vitamin deficiency. Since thiamine, riboflavin, and niacin may act as coenzymes, the above data would support the concept that neuronitis is due to a disturbance of the enzyme system.

The most significant therapeutic fact demonstrated by this study is the value of the respirator in cases in which there is respiratory difficulty.[11] Early in the development of respiratory difficulty there may be no subjective distress. The chief signs are wakefulness, restlessness, fatigue, anxiety, and mental confusion. The patient will use accessory muscles of respiration in the neck and abdomen. Cyanosis is a late stage and should not be allowed to develop. In general, if there is a drop of the vital capacity to 30% of normal, the patient should be placed in a respirator. Tracheotomy, which was used in six of our eight cases, is of value, since it provides an adequate, direct airway and allows satisfactory suction of the lower respiratory tract. It should be used if the patient is unable to clear the oropharynx of secretions by coughing or swallowing, if there is laryngeal obstruction with abductor paralysis, or if there are rapidly progressing signs of bulbar paralysis. With regard to the enzyme system, oxygen is necessary for normal tissue metabolism, and any degree of anoxia due to inadequate ventilation may also contribute to the symptomatology of neuronitis.

We feel that the best therapeutic routine consists of parenteral administration of 50 to 100 mg. of thiamine daily and 1 cc. of crude liver extract three times weekly in combination with dimercaprol, 2.5 mg. per kilogram of body weight every six hours. Administration of vitamins should be continued during the illness, but dosage

10. Chusid, J. G., and Marquardt, G. H.: Acute Infectious Polyneuritis (Guillain-Barré Type), Ann. Int. Med. 23:852 (Nov.) 1945.

11. Austin, E., and others: The Use of a Ventilation Meter in the Evaluation of the Poliomyelitis Patient with Respiratory Difficulty, Phys. Therapy Rev. 32:348 (July) 1952.

should be reduced as the patient improves. Dimercaprol should be given for at least four to six days in moderately severe cases and administration may be discontinued if no improvement is shown. In severe cases, therapy with dimercaprol should probably be continued, sometimes in modified dosage, until the patient has improved materially. It should be discontinued immediately if there is any untoward reaction to the drug. This routine, with the prompt use of tracheotomy and/or the respirator, combined with assiduous nursing care, gives the patient with infectious neuronitis a very good chance for survival even when he has severe bulbar paralysis.

SUMMARY AND CONCLUSIONS

Infectious neuronitis, or Guillain-Barré syndrome, is probably not a disease but a symptom-complex. The main causative factor is not known, but evidence is presented to suggest that it is due to a disturbance of the enzyme systems of the neurons. It is suggested that the administration of dimercaprol in combination with thiamine and crude liver extract is indicated. The prompt and adequate use of the respirator and tracheotomy when indicated will enable the majority of patients with symptoms of bulbar and respiratory paralysis to survive.

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THE FAMILY AS A RESERVOIR OF CHILDHOOD INFECTIONS

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All physicians engaged in the general practice of medicine have observed many instances of family outbreaks of infections and have experienced them in their own family groups. Many of these illnesses are virus diseases. They include family outbreaks of a common cold, virus enteritis with vomiting or diarrhea, or both, as well as a large number of nonbacterial respiratory infections that are classified as tracheal bronchitis not caused by bacteria. These virus infections in family outbreaks have certain things in common; they are usually self-limited and devoid of serious complications, and, unfortunately, there is no specific therapy to be recommended.

Not all family infections, however, are caused by viruses. Many are caused by specific pathogenic bacteria, and these present a variety of difficult and puzzling problems of management to the physician and to the family as well. Perhaps the most important of these bacterial pathogens, which are carried back and forth in the family group and in which the family acts as a reservoir of repeated childhood infections, are those caused by beta hemolytic Streptococcus, hemolytic Staphylococcus aureus, pneumococcus, and Hemophilus influenza organisms.

In the past year I have seen 48 family outbreaks of pneumococcic infection in which two or more members of the family were shown to be affected by the same type of pneumococcus. There was a striking variety of types of disease produced by a single pathogen in these family members. Another important factor was the fact that treatment and improvement of a single member did not solve the problem, because reinfection invariably occurred from a "healthy" carrier member in the family. It is clear that the family physician is in a unique position to realize the familial nature of reinfection in these bacterial diseases and to attack the problem by treating the entire family.

The family group represented in figure 1 consisted of five children, the parents, and an uncle. The first patient seen was a 13-month-old boy with otitis media who had a fever of 104 F. His 5-year-old sister also had a fever—of 101 F—and tonsillitis without exudate. The mother who brought these children to our clinic had no complaints. Nose and throat culture of the two children and the mother revealed pneumococcus type 3 in an almost pure culture. Oxytetracycline ("terramycin") therapy was started for the two children and discontinued after four days of treatment, following a very satisfactory clinical response. Nose and throat culture was repeated, and both children had a negative culture. A culture was made for the untreated mother again, and she was still found to be a carrier of pneumococci type 3.

Two weeks later, the 13-month-old boy was seen again with otitis media and fever, and the mother complained of a postnasal drip and sinus pain. She had no fever. She brought the 5-year-old girl, who had been previously treated, as well as her 3-year-old girl who had not previously been seen, and who had a vaginal discharge. Nose and throat culture again revealed type 3 pneumococci in the 13-month-old child. The 3-year-old girl, who had vaginitis, had a pure culture of pneumococcus type 3 in her vaginal discharge. Culture from the previously treated 5-year-old girl was again positive for type 3 pneumococcus in the absence of any disease. On request, the mother brought in the remainder of the family for culture. The uncle, who had complained of a sore throat but had no fever, the father, the mother, a 12-year-old brother, and an 8-year-old girl all carried pneumococci type 3. The father also had pneumococci type 5 in his nose culture. In the 3-year-old girl a mild case of peritonitis developed subsequently, which responded to oxytetracycline therapy. In view of this generalized involvement of the entire family, all were treated, except the father, who refused to take the drug for reasons of his own. Five weeks later, the 13-month-old boy was again seen with a high fever and appeared extremely toxic. His white blood cell count was 35,000, and bacteremia and septicemia were suspected. Blood culture was positive for pneumococcus type 5, which had previously been found in the father's nose and throat culture. Cultures were made again for

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the entire family, and, with the exception of the uncle and the 5-year-old girl, all were positive for pneumococcus type 5.

It is most interesting that the pneumococcus organism in this family was able to cause fever, otitis, sinusitis, pharyngitis, bacteremia, vaginitis, and peritonitis—a variety of conditions that might not ordinarily be associated with a single pathogenic organism. In addition to these types of diseases, I have seen pneumococci meningitis, pneumococcic conjunctivitis, and pneumococcic pneumonia in other families.

The hemolytic Staphylococcus aureus, which is a causative organism in a variety of purulent skin infections, has also played havoc in a family group. Because of the increasingly great number of penicillin-resistant staphylococci, therapy and total eradication has become more difficult as time has gone on. Figure 2 shows a family consisting of a newborn baby, mother, father, grandmother, a brother, and a sister who sometimes handled the infant. I am not sure where the infection originated, but it was first manifested by a superficial nipple abscess in the mother and impetigo of the face in the child. A culture of hemolytic Staphylococcus aureus was obtained, and the infant was treated with local therapy. The mother was treated with aureomycin with good response. Shortly after being returned home, the child had seborrhea of the scalp with scaling, which was positive for Staphylococcus aureus. It was noted that the sister who handled the infant had boils on her hand and arm, which also were positive for Staphylococcus aureus. She was not treated and improved spontaneously. The child's skin infection became worse and typical of impetigo of the face. A massive breast abscess requiring penicillin treatment and drainage in the hospital promptly developed in the mother. Staphylococcus

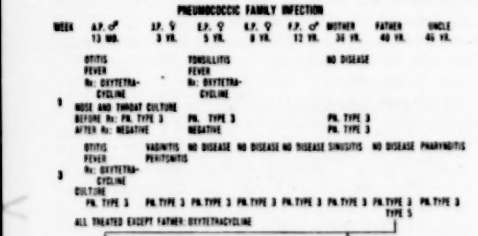

Fig. 1.—Course of infection by pneumococci in one family.

aureus was cultured in both the infant's scaling lesions of the face and the mother's pus. The grandmother, during the mother's hospitalization, had been "burping" the child by holding him against her left cheek, and boils developed on that portion of the face and her left ear—the latter infection being most intractable to penicillin, sulfonamide, and aureomycin therapy. The child was hospitalized for two weeks for intensive aureomycin therapy and improved markedly. The mother improved and was discharged from the hospital, but the sister still had some boils on her hands and arms. The child returned home, seemed to do well for two weeks, and then massive impetigo developed on the face and body. At the same time boils developed on the hand and arms of

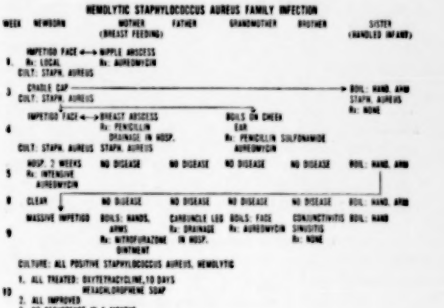

Fig. 2.—Course of infection by Staph. aureus in one family.

the mother, which responded to nitrofurazone (furacin®) ointment. In the father at this time a carbuncle of the leg developed and required drainage in the hospital and a fairly prolonged stay. The grandmother had a flare-up of the boils on her face, requiring aureomycin therapy. Conjunctivitis and sinusitis due to Staphylococcus aureus developed in the brother, for which no treatment was given. The sister still had a boil on her hand. On the 10th week the entire picture, which had previously involved three different physicians, was brought together. In consultation with the others, one of the physicians decided to treat the entire family with the drug that seemed to be most effective as judged by sensitivity tests. Marked improvement resulted when the entire family was treated with hexachlorophene (gamophen®) soap and oxytetracycline for a period of 10 days.

This family had skin infections of all kinds, conjunctivitis, sinusitis, and three hospital admissions requiring a great deal of outlay in time and expense. If this family had had one physician instead of three, they would very likely have done better because earlier treatment of the entire family would undoubtedly have been instituted.

The beta hemolytic Streptococcus is another pathogen that can be harbored in the family and usually causes family reinfections to occur. In the instance shown in figure 3 an 8-year-old girl was seen with fever and tonsillitis. She was given sulfisoxazole (gantrisin®) by mouth, with good clinical response. In her 1-year-old brother otitis media developed, and he was given penicillin for three days. The 12-year-old sister, who had had rheumatic fever at the age of 9 years, began to have fever and cervical adenitis and was given sulfisoxazole. The father had a sore throat without fever and was not treated, while paronychia of the left thumb developed in the maid and was not treated. Nose and throat cultures were done on the entire family a few days later. At that time, all of them had group A beta hemolytic Streptococcus type 12 in the cultures.

Since they were all clinically improved, and before the culture report was received, the family went on vacation. During the early portion of the vacation period the 12-year-old girl experienced reactivation of her rheumatic fever, with arthralgia, gallop rhythm, and high fever. She was hospitalized and given penicillin. One week later the 8-year-old girl again had tonsillitis and fever with arthralgia, and a tentative diagnosis of rheumatic fever was made. She was given penicillin intramuscularly at home. At nine weeks specimens from the entire family were recultured. The 1-year-old boy had a negative culture, probably having had a barely sufficient amount of penicillin. The 4-year-old boy, who never had been sick, was still a carrier of beta streptococci. The 8-year-old girl, having received penicillin intramuscularly for a week, had a negative culture as did the 12-year-old girl, who was then released from

Fig. 3.—Course of infection by beta hemolytic streptococci in one family.

the hospital. The culture from the untreated mother, who previously had been a carrier, had become negative spontaneously. The culture from the father, who had not been treated and who had previously been a carrier, still remained positive. The culture from the maid, who previously had an infected finger and a positive throat culture, had spontaneously become negative. The entire family was treated with penicillin intramuscularly in four doses, 36 hours apart. Cultures after therapy were negative and remained so for a three month period thereafter. Otitis, tonsillitis, adenitis, paronychia, and rheumatic fever were present in this family group, and reinfection from carrier members was a prominent finding.

COMMENT

I have shown examples of the family as a reservoir of bacterial infection of a quite spectacular nature. For each example, there are many cases in which only one or two members are involved, usually in such a way that a given child repeatedly gets infected from an adult carrier. Occasionally the opposite happens. In addition to the three bacterial pathogens that have been described, all the enteric pathogens such as those of the Salmonella and Shigella group, of course, travel in families. There again, eradication of all carriers is essential in the successful management of a case. It will be noted that, with the exception of skin and enteric infections, almost all such family infections are respiratory in nature. It is difficult to say in a given instance whether a respiratory disease is of bacterial or viral etiology without either bacteriological cultures or fairly involved serologic tests. It is obviously not economical in the average case in daily practice to resort to these techniques very frequently. The diagnosis of bacterial versus viral infection frequently rests on clinical judgment, the degree and type of fever, and perhaps the presence of an elevated or a depressed white blood cell count.

It is no wonder that such a family reservoir is so frequently present in childhood infections. The great intimacy of family contact predisposes to repeated and massive exposure of a susceptible family member to a carrier whom he sees, frequently touches, and kisses day in and day out. To physicians in family practice, this subject offers a unique challenge and allows for a fine contribution to family health. There is no doubt that intensive exposure, even in the face of low pathogenicity, is overwhelming in family infections.

The family in figure 3 illustrates the importance of the family reservoir of bacterial infections in illnesses in which repeated infections with the beta Streptococcus are particularly undesirable. These include rheumatic fever, glomerulonephritis, and nephrosis. Not infrequently, when a child who is getting better in a convalescent home returns to his own family, he is again the victim of reinfection from the original family source. One would not be amiss in checking the family for the presence of beta streptococci before such a child returns to his family.

The family physician is best able to assess the nature of the family as a reservoir of repeated bacterial infections. He alone is able to treat each and every member of the family if he deems it desirable. Besides the cost of antibiotic therapy, such early "family therapy" may save needless illness as well as a good deal of money in the long run. Recognition of the family as a reservoir of bacterial infection is a responsibility of all those dealing with family health.

SUMMARY

In summary, it has been the purpose of this paper to point out the frequency and nature of a number of bacterial infections rampant in a given family. Stress has been placed on the variety of clinical diagnoses one might find, despite the fact that a single pathogen might be causing all disease. Recognition of family infection and treatment of the entire family, including those who are not sick, with the best antibiotics at hand ostensibly offers the best hope for the management and prevention of repeated reinfection from the family source of children who are chronically ill.

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ANALYSIS OF THE VALUE OF CHEMOTHERAPEUTIC AGENTS IN TREATMENT OF TUBERCULOSIS

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The unlimited possibilities of chemistry make it likely that eventually a chemical cure for tuberculosis will be found. Many a premature outburst based on empiric or clinical findings will result, however, from improper or "forced" test (screening) methods that lack a scientific basis and show results that cannot be translated directly into fundamentally sound information. Every newly discovered "cure" for tuberculosis [1] has brought its subsequent unsolved problems of dubious scientific value that required laborious experimentation of the kind that exhausts the meager funds allotted to the sincere scientific investigator. Tuberculin therapy of the nineties was announced with the éclat of a new world war and required a half century to fade from use. Moellgaard's gold cyanide therapy is still considered worth while by authors of standing, although Knopf and Amberson early refuted its value. So, too, some of the more recent chemotherapeutic agents for tuberculosis such as chaulmoogra oil, the sulfonamides, glucosulfone (promin®) sulfoxone (diasone®), para-aminosalicylic acid, the semicarbazones (thiomithiozone, amithiozone), and others still find favor with certain clinicians because they believe the old favorable empiric reports and ignore subsequent scientific evidence.[2] It seems that the grasp for the new beclouds for some time the painstakingly gained negative evidence that must follow in the wake of most explosive new empiric discoveries.

In view of the ever increasing number of new chemotherapeutic agents and antibiotics used in treatment of tuberculosis, this report cannot possibly be all-encompassing. In addition to the numerous sulfonamides used in the last decade and para-aminosalicylic acid, now available are the streptomycins, including neomycin and oxytetracycline ("terramycin"), the numerous thiosemicarbazones, and the more recent isoniazid compounds, with which it is claimed the patients are "up and dancing" within a short time because they are "cured" by inference. Thus even deserved merit may be beclouded by over-zealous exploitation based on premature enthusiasm resulting from too speedy methods of testing.

An analysis of the value of chemotherapeutic agents in the treatment of tuberculosis must concern three fundamentally important issues: first, an understanding of the tubercle bacillus and how these agents actually affect this micro-organism; second, an understanding of the action of these agents in vivo as well as the action of the tubercle bacilli in vivo; and, third, the ways by which the body can circumvent action or even lead to the assumption by the observer of direct therapeutic action that may not exist. Since only a few chemotherapeutic agents have generally acknowledged therapeutic action in tuberculosis and since understanding of the the mechanism of their in vivo action is still vague, this report is concerned only with some known facts that can be guides in interpretation of the action attainable with these chemotherapeutic agents. In such an analysis it is natural, first, to consider the tubercle bacillus from all possible angles pertinent to the action of a therapeutic agent and, second, to determine criteria for an agent's ability to fulfill the requirements of a cure or *magna sterilisans* (Ehrlich) that appear attainable but which previous agents have completely failed to achieve.

It is well to realize that man has no doubt succeeded in obliterating the tubercle bacillus in vivo (bacteriologically) in exceptional cases, and so there is still hope that this result can be achieved by direct action of a chemical agent. Thus far there is no proof that any agent known to date by itself can eliminate the bacillus within a reasonable time within the body or under well-controlled conditions in active concentrations that are attainable in the body in vitro. It is only necessary to refer to the difficulty of maintaining viable tubercle bacilli in vitro to realize the ease with which the biological properties of the bacilli can be misinterpreted. While keeping bacilli alive in the laboratory for a year was a pronounced accomplishment in the twenties, today records disclose that these bacilli can remain viable for over 30 years in certain mediums [3] and that even dehydrated bacilli can yield new growths after five years. So also new concepts have arisen in regard to the growing of the mammalian tubercle bacilli, and today it is acknowledged that the mammalian bacilli can grow intermittently without apparent detriment and at temperatures well below that of the mammalian body.[4] Even the problem of the nature of virulence of the mammalian tubercle bacilli has only been touched on, without much actual information being accumulated as to the cause of transition, although we are aware that the bacilli can become avirulent within or outside the host.[5]

By means of certain dispersing agents, it has been possible to obtain a claimed speeding of growth of diffuse subsurface liquid cultures of mammalian tubercle bacilli,[6] but it is doubted whether actual multiplication has been speeded and whether the phenomenon is not really one of better visual appreciation due to multiple plants rather than of actually increasing the natural rates of development. It is singularly interesting that the mammalian tubercle bacilli can grow on the simplest synthetic mediums under certain conditions and can utilize in small plants the most complicated organic colloidal body constituents for growth.

Pertinent to this is an experiment on the requirements of the tubercle bacillus for simple inorganic constitu-

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ents, on which the literature is rather vague [7] because most of the earlier studies were pursued before purified inorganic chemicals were available. These results are of a study in which a simple synthetic medium (Wong-Weinzirl's) was varied as to salt constituents. The study was an outgrowth of an observation with streptomycin-sensitive and streptomycin-resistant human tubercle bacilli of the same strain that were planted on the synthetic medium. The synthetic medium was free of magnesium sulfate but varying amounts of streptomycin sulfate or streptomycin calcium chloride complex had been added. While the streptomycin sulfate supported good growth, the streptomycin calcium chloride complex failed to do so even with highly resistant bacilli. The results of a relatively short (six weeks at 37 C) growth experiment with added salts is recorded in the table.

Growth of Human Tubercle Bacilli on a Simple Synthetic Nonprotein Medium Free from Magnesium, Calcium, and Sulfate After Six Weeks at 37 C*

Salt Added	Gram per Liter of Salt Added†				
	0	0.001	0.01	0.1	1.0
$MgSO_4$	±	±	+	+++	+++
$MgCl_2$	±	±	±	±	+
Na_2SO_4	±	±	+	++	++
$CaCl_2$	±	±	±	±	±
Streptomycin SO_4‡	±	±	±	++	++
Streptomycin $CaCl_2$‡ complex	±	±	±	±	±

* Wong-Weinzirl's nonprotein synthetic medium consists of monopotassium phosphate (6 gm.), magnesium sulfate (1 gm.), ammonium citrate (5 gm.), sodium carbonate (2 gm.), malic acid (3 gm.), glycerol (50 gm.), ferric ammonium citrate (0.05 gm.), sodium chloride (1 gm.), and 1% dextrose in 1 liter brought to pH 6.8 with ammonium hydroxide. The magnesium sulfate and sodium chloride were omitted from the medium in this study.

† The amount of growth of the bacilli is recorded arbitrarily from 0 representing no growth to +++ representing a good normal surface-covering growth during the time of incubation. Apparent doubtful growth is indicated by ±.

‡ The results recorded with streptomycin are for the same strain of human tubercle bacilli resistant to streptomycin.

The results recorded in the table make it obvious that retardations in growth of human tubercle bacilli can be tempered by slight modifications in the amount of certain inorganic radicals (for example, the sulfate radical, SO_4), and no doubt the amount of more complex active organic radicals,[8] which can, under certain circumstances of chemical reactions, be inactivated as essentials to growth. Also, magnesium has been noted to stimulate growth appreciably. These facts are pertinent when it is realized that retardant effects are used as criteria of the action of antibiotic and chemotherapeutic agents in vitro, effects which are greatly obliterated or reduced in the more complex colloid organic mediums such as egg mediums.[9]

During the early experimental periods with streptomycin and with the flood of experimental evidence to indicate its apparent phenomenal action against the mammalian tubercle bacillus, particularly in the highly susceptible guinea pig, it was difficult not to conclude that here was an agent that in itself was destroying the tubercle bacillus in vivo. It definitely and strikingly prolonged life, and so it was concluded by some investigators that it was destroying the tubercle bacilli.[10] Yet in vitro in a neutral phosphate buffer at 37 C, the tubercle bacilli remained alive in contact with 1,000 and 10,000 units of streptomycin (or dihydrostreptomycin) per milliliter for several years, whereas the body concentration following injection rarely exceeds 10 μg (units) per milliliter for any time.[11]

This led to the use of the intravenous (ear vein) lethal infection test,[12] from which it was noted that streptomycin in large doses in the guinea pig for as long a time as possible was unable to prevent death from intravenous infection with virulent human tubercle bacilli, even though the life duration was extended from an average of 20 days to 200 days.[11] In terms of human therapy, however, it is obvious that it could not be expected that streptomycin per se could actually destroy the human bacilli in vivo even though its temporary retardant action could be significant under certain circumstances. Although during this period of 200 days of streptomycin therapy, resistance could materially increase, it was not clear that this was the entire explanation of the failure to maintain the life of the guina pigs infected with lethal doses intravenously.

DEVELOPMENT OF RESISTANCE

Since this report considers chemotherapy from the standpoint of the tubercle bacillus, it may be well to consider the still vaguely understood phenomenon of resistance to chemotherapeutic agents. This resistance now appears to be an almost universal phenomenon, since it exists for most of the sulfonamides, the antibiotics, streptomycin, dihydrostreptomycin, neomycin, the semicarbazones and para-aminosalicylic acid, and from recent reports it appears to be established and is being admitted for the isoniazid compounds. Since the work with streptomycin has advanced beyond the more speculative stage, there are still voids in knowledge as to the mode of action or the mode of development of resistance. In my own experience adaptation of the mammalian tubercle bacilli to streptomycin has seemed to be responsible rather than biogenetic selection.[13] The reasons are that there is a relation between the drug concentration and the time such resistance develops in vitro and in vivo and that resistance can develop not only by mere contact without evident multiplication of the bacilli, particularly in vitro in non-nutrient solutions but also in nutrient solutions and in a non-nutrient neutral phosphate buffer solution.[14] In the phosphate buffer solution, the concentration of the antibiotic, temperature, and time are particularly pertinent; in highly nutrient mediums, certain obvious irregularities are evident, but the change can occur quite speedily and in one transfer. Yet in twilight zone tests with varying mixtures of streptomycin-sensitive and streptomycin-resistant human tubercle bacilli, animal treatment tests did not disclose the presence of small numbers of resistant forms among pure cultures of nonresistant forms of human tubercle bacilli.[13]

On the basis of these observations and therapeutic tests in animals, it was advocated that treatment with streptomycin could be just as effective with moderate intermittent doses as with the large toxic doses [15] and to

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the decided advantage of the patient—a method now universally conceded for most cases. Although there is a decided trend to use of combinations of therapeutic agents to avoid development of resistance to antibiotics, it would appear to involve inherent toxic dangers from prolonged use not particularly to the advantage of the patient. It can only be a method of acknowledging the futility of chemotherapy at present and the grasp for something that resembles the last cycle of treatment of tuberculosis—the era of small doses of tuberculin and the "gun shot" prescription.

If it is admitted that the mammalian tubercle bacillus possesses striking ability to survive outside and inside the body and an adaptability for survival quite striking for a pathogen [16] and that this micro-organism develops resistance to all types of chemical and antibiotic retardant agents, there will be no hesitation in accepting therapeutic aid from these agents but not to the point of proclaiming bacillicidal effects that have as yet not been achieved. It will not dampen the ardor of scientific endeavors to continue the search for a chemotherapeutic agent that will kill tubercle bacilli, and respect will not be lost for the mammalian tubercle bacillus. It is obvious that the criterion for a potent chemotherapeutic agent for tuberculosis must be the ability to destroy the bacilli efficiently in a susceptible animal host to such an extent that the resistance and specific immunity developed during the course of treatment tests will not persuade the observer that more is being achieved with the agent than could be achieved without it.

In critical evaluation of the recently advocated isoniazid compounds, the most that can be said, until it is proved otherwise, is that they can only be considered another set of chemicals that act as retardants, to which the tubercle bacilli can develop a decided resistance, and that are incapable in themselves of curing tuberculosis. When administration of these compounds has been discontinued, the tubercle bacillus has not been bacteriologically eliminated except possibly insofar as natural processes of immunity are concerned, which is still unmeasurable by any direct means. They are useful in acute episodes, although in only limited numbers of such episodes, and so will aid in the patients' welfare and ultimate recovery. They should not be abused where successful progress is possible without them but should be conserved for use in cases of urgent necessity. They should not be wastefully exhausted of their later value.

The isoniazid compounds (variously named rimifon,* hydrazid,* and marsilid*) are now being studied. It has been reported that "these drugs have shown remarkable activity against the tubercle bacillus in the test tube, in the experimental animal, and in preliminary tests in man, but no final statement can yet be made as to the optimum dosage schedule, the permanency of the early results obtained, or the possibility of drug resistance." This is quoted from a recent conservative editorial [17] that summarizes the reports and concludes, "It is evident that this group of compounds has several advantages over other antituberculosis agents." Yet the figures and data are not critically analyzed as to methods or results and possibilities. Likewise, the first publications failed to be entirely convincing that these compounds would outstrip others in use except in possible ease of production. Even though much information should be forthcoming, a few definite facts do warrant conservatism in their use. In the first animal reports,[18] based on a sketchy 21 day treatment mouse test, it was maintained that "practically all animals were free of viable tubercle bacilli after the twenty-first day of treatment with 10.0 mg./kg. Rimifon . . . and in case of Marsilid the same effect was achieved with 25.0 mg./kg." All possibilities were apparently not realized then, such as changes wrought by the drug on the distribution of the bacilli in vivo,[19] on the cultural results, on the medium, and on their immediate multiplication in mediums. This is obvious from the fact that it is known that unless the deposition of bacilli following intravenous infection is altered, the tubercle from the initial large infection dose used should produce tubercles through mere mechanical and chemical presence. Cautious, in regard to cultures, is the statement, "A more definite statement as to whether the lack of growth in the cultures is an indication of irreversible damage to the tubercle bacilli must await further experiments with more sensitive methods," which suggests that the authors are doubtful of their own results. This also applies to the tests in which action was greater against the supposedly milder disease resulting from intranasal infection compared with intravenous infection. At the time of writing,[20] the clinical reports of symptoms, possibly justifiedly, are enthusiastic but misleading bacteriologically (smear test), "noting conversion of the sputum to negative" without adequate proof or time for such proof. Aiding such over-enthusiasm was the cure (with specimens negative for tubercle bacilli) of one Macaca rhesus monkey.[21] Toxicity has been acknowledged in animals [22] studied more carefully. A subsequent article [23] on mouse infection extended the observation period for 21 days without treatment, cited comparative tests with other agents such as streptomycin, *p*-aminosalicylic acid, amithiozone, and others purporting to allow tubercle formation in contrast, with the conclusion that the isoniazid compounds act by a "direct effect on the viability of the causative organisms." A more revealing clinical article [24] discloses that unconcentrated sputum smears in 25% of patients became negative (which may have been a consequence of reduced volume or excretion to a great extent) and that roentgenographic changes were not of crucial significance in the cases treated. Symptomatic changes (return of appetite, decrease in temperature, and gain in weight) were dramatic. In a study of blood levels of isoniazid in dogs and humans,[25] a peak level of less than 0.7 mg. per 100 ml. of plasma was obtainable. Plasma concentration in dogs 30 minutes after feeding could rise to as high as about 10 μg per milliliter with lethal doses [26]; while in humans receiving 3 mg. of isoniazid per kilogram of body weight, a maximum of 3 μg per milliliter was achieved in one case, with the average about 1.0 μg at six hours and 0.4 μg in 24 hours. With isoniazid administered in two doses a day, levels of 0.6 to 1.5 were attainable.[27] A suggestion of the eventual proper cataloguing of the effects of isoniazid appears in an article reporting studies in the Trudeau laboratory [28] in which speedy methods and mediums were used, with

the result that static effects for iproniazid (with levels of 1.6 to 6.2 μg per milliliter) and isoniazid (with levels of 0.025 to 0.05 μg per milliliter) are recorded. It is stated that iproniazid destroys (reduces) tubercle bacilli from 200,000 to 2,500,000 (in control growth) organisms (in tween* albumin medium at 37 C) to <10 in a 10 μg per milliliter concentration in five days. An addendum, in small type, adds, "When incubation of the inoculated tubes of tween-albumin medium containing isonicotinic acid hydrazide was continued for 5 weeks, growth eventually appeared in concentrations as high as 25 μg per ml. It has been demonstrated that this delayed growth consists of tubercle bacilli which have an increased resistance to the drug." Pointedly, it is evident that isoniazid cannot destroy the human tubercle bacillus in appropriately controlled experiments in concentrations of from 25 to 100 μg per milliliter within a reasonable time of two to three months at 37 C and that, likewise, it does not destroy them per se in appropriately controlled animal tests in concentrations obtainable in vivo in man or animals and thus cannot be catalogued as bacillicidal in vivo in the concentrations attainable. In other words, it is not a "cure" per se. Resistance of human tubercle bacilli to isoniazid develops definitely within a variable time in vitro and apparently in vivo, as it does for streptomycin. Experimentally then, treatment is of doubtful value.

In agreement with the report from Trudeau, it seems that isoniazid is superior to any other antituberculosis agent except streptomycin in experimental tuberculosis and in its action on the tubercle bacillus. It is not to be inferred that these agents cannot find a place in the treatment of tuberculosis, but both are limited in value; they are only temporary retardants or static agents, because the tubercle bacillus possesses the ability to build up a complete resistance to both of them individually that lasts for years. Therefore, they may be used to complement each other in therapeutic use, another favorable feature that has been claimed for a number of others unconvincingly in the past. They should not be used except with the fullest knowledge of their limitations and with a view to the best possible values; they should not be used promiscuously in every case of tuberculosis. Thus they can be valuable adjuvants to the regular regimen of tuberculosis therapy but do not appear to possess the power of assuring the universal abolition of tuberculosis. Their discovery encourages unhampered continuous search for a true efficient in vivo bacillicidal agent. This search should be based on careful long-term animal tests rather than on purported speed schemes devised mainly for mass screening purposes and thus resulting in error and exaggeration by inference. The tubercle bacillus does not reveal all its intimate protective characteristics speedily, if past experience is significant, and retractions are wasteful and ultimately harmful and time-consuming to investigators and patients alike. Haste so far has made waste in the combatting of the tubercle bacillus.

SUMMARY AND CONCLUSIONS

An analysis of chemotherapeutic agents in the treatment of tuberculosis resolves itself into an appreciation of the following facts:

1. The tubercle bacillus is an exceptional micro-organism among pathogens in that it is capable of surviving under the most adverse biological conditions, including (*a*) over 30 years at incubator (37 C) temperature; (*b*) over 5 years dehydrated and sealed in glass; and (*c*) in the body for decades, at least in a virulent or low virulent state in the absence of evident growth. 2. In spite of this survival capability, the bacilli apparently can be destroyed slowly in the body under, to them, unfavorable natural biological conditions. 3. The bacilli can grow in intermittent stages of favorable conditions on nutrients and in the body for years. 4. Growth (static) and destruction (bacillicidal) laboratory tests on the bacilli must be evaluated guardedly and cannot be judged speedily since the bacilli are capable of adapting themselves to most antibiotics and chemotherapeutic agents within a relatively short time in vitro and in vivo. 5. At present, according to carefully controlled and evaluated studies, two chemotherapeutic agents—the antibiotic streptomycin (and its closely related compounds) and isoniazid (and its homologues)—possess merit as static adjuvants to the scientific medical treatment of tuberculosis for limited periods of time only and under the best medical supervision because the bacilli develop a resistance to their retardant action. They also can be used to supplement each other for this same reason. They are, however, not in themselves "cures" for tuberculosis, since they have not been demonstrated to destroy the tubercle bacillus in vivo efficiently or within a reasonable time. 6. The discovery of these agents, a notable advance, should encourage the unhampered scientific search for more efficient static agents and an in vivo bacillicidal agent against the tubercle bacillus.

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Overspecialization.—Every scientist feels acutely today the effects of overspecialization. The volume of publication is so vast that it is impossible to keep abreast of it, even as the field of interest is narrowed. Whole new sciences and branches of engineering appear, with their specialized societies and journals. Intensely progressive gatherings of research workers develop their own jargon, unintelligible except to the initiated, heightening the barriers which separate their works from the main stream of progress.

We are told that, long ago, there was a strange sort of real estate development. My memory of what I learned about it in my youth is somewhat hazy. But it seems that there was a project to construct some sort of Empire State Building, only much larger and more grandiose, reaching up toward the sky and mingling with the clouds. It was called the Tower of Babel. As the story goes, it was never completed, for the plans ended in confusion. The workmen suddenly found that each one was speaking a special language and that no man understood what the other was saying. So the minor construction went on piece by piece, but none of the pieces fitted; and the general plan of construction was completely lost. We are in danger, in science, of building a Tower of Babel. This is especially true in the science that deals with man. For man is complex, with a complexity far exceeding that which was assumed in the early days when the practice of medicine was a simple matter of applying empirical experience and the science of medicine was nonexistent. As we have learned more, so have we learned that there is vastly more to learn before any comprehensive over-all understanding can emerge.—Vannevar Bush, Eng.D., "Science in Medicine and Related Fields," *Medical Annals of the District of Columbia*, January, 1953.

PERIPHERAL VASCULAR FAILURE AS CAUSE OF DEATH IN GENERALIZED EXFOLIATIVE DERMATITIS

REPORT OF FOUR CASES

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Death in skin diseases occurs usually from infection, septicemia, toxemia, inanition, and exhaustion; in the case of systematization of the disease, involvement of vital organs is sometimes fatal. LeVan and Sternberg,[1] in 1949, listed, as immediate causes of death in a series of 120 cases of skin diseases, bronchopneumonia, congestive failure, coronary accidents, uremia, lobar pneumonia, meningitis, the skin disease per se, and "miscellaneous" conditions. In 15 fatal cases of generalized exfoliative dermatitis, 8 of the patients died of bronchopneumonia, 3 of cardiac failure, 1 of lobar pneumonia, and 1 of anemia, and in 2 the dermatitis was reported as the cause of death.

In contrast to cardiac failure, peripheral circulatory collapse seems rarely to be the cause of death in exfoliative dermatitis or in skin diseases in general. In some reports there are vague suggestions of the presence of shock. Particularly in generalized exfoliative drug dermatitis [2] and in the sudden deaths of eczematous infants (probably due to overwhelming infection),[3] a collapse of the peripheral circulation seems frequently to be the immediate cause of death. Shock is also, as is generally known, the usual mechanism of early mortality in severe extensive burns. However, in other fatal processes involving large skin areas, e. g., in pemphigus and in most types of generalized exfoliative dermatitis except drug dermatitis, peripheral circulatory collapse has not been noted in the literature. This type of death, however, is probably more frequent than is apparent from the paucity of reports.

In this paper, four fatal cases of generalized exfoliative dermatitis are reported, and we wish to call particular attention to peripheral circulatory collapse as the cause of death. At the same time, it is our intention to discuss certain endocrine and infective factors responsible for the failure of the peripheral circulation. In three of the four cases, autopsy showed abnormal changes in the adrenal cortex. In two of these cases it appeared possible that cortisone and corticotropin were involved in the causation of the fatal shock state. Infections of the skin were present in all four cases and infections in internal organs in at least three of them.

REPORT OF CASES

CASE 1.—A 31-year-old white man had had a dermatitis of the genitalia accompanied by conjunctivitis, coryza, and arthritis, in 1938, and a dermatitis of the antecubital fossae and wrists every winter since 1945. Early in 1950, pruritus ani developed for which the patient treated himself with diothane® (diperodon with hydroxyquinoline benzoate), cyclomethycaine (surfacaine®) hydrochloride, and salicylic acid and coal tar ointments. A generalized dermatitis of contact type resulted, and the patient was admitted to the Brooklyn Veterans Administration Hospital on Aug. 20, 1950.

At the time of admission the patient had a generalized exfoliative dermatitis with some lichenification in the antecubital fossae and diffuse weeping and crusting in the genital and anal regions and the lower extremities. There was bilateral inguinal lymphadenopathy. The temperature was 101 F, the pulse rate 90, and the blood pressure 110/70. The red blood cell count was normal, the hemoglobin between 11.7 and 13.2 gm. per 100 cc., and the leukocyte count ranged from 10,900 to 26,000 per cubic millimeter. Urine examination revealed hyaline casts and cylindroids. Roentgenograms of the chest and serologic tests for syphilis were normal.

With bland local applications, antihistaminics, and sedation there was improvement within 10 days, and almost complete recovery in one month. At that time patch tests with the above-mentioned ointments and with contraceptive rubber gave strongly positive reactions, especially to cyclomethycaine hydrochloride. Within 24 hours there was a generalized flare-up for which cortisone was prescribed for 10 days in daily doses of 100 mg. Hematocrit and blood chemistry values, including those for sodium, potassium, and chlorides, and the serum proteins were normal at that time and remained so; however, the Thorn test showed a paradoxical increase of 19% of eosinophils after three hours and a 35% increase after four hours. There was, nevertheless, significant improvement with cortisone therapy. A severe relapse followed immediately after discontinuation of this drug. Ten days later the cortisone injections were resumed, but after two days the patient complained of nausea, vertigo, and weakness. He began to vomit, and he had fever. This time the dermatitis did not respond to therapy. On the following day the blood pressure dropped to 70-80/40-50. Cortisone therapy was discontinued, and antibiotics, parenterally administered fluids, arterenol, desoxycorticosterone (doca®) acetate, lipid adrenocortical extracts, cardiac stimulants, and oxygen were used for about three days, but the peripheral failure could not be controlled, the blood pressure rising to 80-90/40-50 at the most and falling at times to 50/30. Finally no pressure could be obtained, and the patient died.

Pertinent autopsy findings included, apart from the severe generalized dermatitis, necrobiosis of adrenocortical cells with increased cytoplasmic granularity and decreased vacuolization. This was regarded as evidence of "stress." The spleen was flabby and friable, and no malpighian corpuscles were discernible; on microscopic examination there was marked congestion of the pulp with many eosinophils. There were also degenerative changes of the liver parenchyma and of the wall of the gallbladder and punctate hemorrhages in the renal pelvis.

CASE 2.—A 42-year-old white man had tender swellings of the anterior cervical lymph nodes in 1944 and cervical and axillary lymphadenopathy in 1947. A biopsy specimen at that time was reported to show giant follicular lymphoblastoma.

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This study was sponsored by the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the authors are a result of their own study and do not necessarily reflect opinion or policy of the Veterans Administration.

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Subsequently, generalized lymphadenopathy developed, including the mediastinal and epigastric lymph nodes, for which the patient received x-ray therapy with good temporary results. Late in 1950, progressive inguinal lymphadenopathy was again treated with x-rays. At that time a mucosal ulceration appeared on the floor of the mouth for which the patient used penicillin lozenges. A few days later a generalized pruritic eruption developed simultaneously with generalized aches and joint pains. There were also at that time axillary and cervical swellings, splenomegaly and hepatomegaly, anemia, and hypoproteinemia. A Thorn test revealed a drop in eosinophils of 27%. The patient was given 200 mg. of cortisone daily for nine days, with some improvement. At this point, on Feb. 7, 1951, he was admitted to the Brooklyn Veterans Administration Hospital.

On admission, the patient appeared seriously ill. He had a generalized, exudative, weeping and crusted exfoliative dermatitis, ulcerations of the lips and of the oral mucosa, enlargement of the anterior cervical and axillary lymph nodes, hepatomegaly, and splenomegaly. His temperature was 100 F (37.7 C). The pulse rate was 96 and the blood pressure 120/60. The red blood cell count and hematocrit were normal, and the hemoglobin was 12.4 gm. per 100 cc. White blood cell and differential counts were within normal limits (until the day of death, when there were 77% neutrophils with 47% band forms). Bone marrow examination showed hyperactivity compatible with chronic infection. Sedimentation rate was 25 mm. per hour. A serologic test for syphilis was negative. A roentgenogram of the chest was normal. Serum proteins totaled 5.69 gm., of which 4.27 gm. was albumin and 1.42 gm. was globulin. Results of urinalyses were normal except on the day of death, when there were 10 to 12 white blood cells per high power field and a 4+ reaction for sugar. A skin biopsy revealed epidermal spongiosis and a massive cutaneous infiltrate of lymphocytes, histiocytes, and macrophages, especially in the papillary bodies.

Treatment was continued with 100 mg. of cortisone administered twice daily for six days, but there was no improvement. The patient was then treated with 25 mg. of corticotropin, administered four times daily, but this treatment also had no effect and was therefore discontinued after three days. In the meantime, the mucosal ulcers became worse, and therefore high doses of aureomycin were given intravenously. Fluids were given parenterally and blood transfusions were also administered, both on frequent occasions. On the 19th hospital day, corticotropin therapy was reinstituted. A single dose of 25 mg. was given at 9 a. m. About three hours later the patient went into shock. There was bile-stained emesis, and the patient became comatose. Arterenol, a second dose of corticotropin (25 mg. administered intravenously), and blood transfusion were of no avail, and the patient died after a few hours in peripheral circulatory collapse. Permission for autopsy was not obtained.

Case 3.—A 40-year-old white man had had a dermatitis of the popliteal fossae, axillas, and groins intermittently since 1943. Late in 1947, after a series of injections of unknown nature, generalized exfoliative dermatitis developed. At the time of hospitalization, the generalized eruption showed areas of denudation, weeping, crusting, fissuring, and marked exfoliation. There was axillary and inguinal lymphadenopathy. The patient was afebrile; his pulse rate was 80 and his blood pressure 124/78. The hematocrit was normal. The red blood cell count was normal, with hemoglobin of 12.1 gm. per 100 cc. The white blood cell count became abnormal two days prior to death, at which time leukocytosis developed, with 17,000 cells per cubic millimeter, of which 80% were neutrophils. At the same time, a reversal of the albumin-globulin ratio occurred. Serum urea nitrogen and chloride values became elevated one day before death. Serologic tests for syphilis were negative, and urinalysis gave normal results. A roentgenogram of the chest was negative. Biopsy of a lymph node showed hyperplasia.

Bland topical treatment, parenterally administered fluids, and penicillin did not change the course of the dermatitis. Over a period of two months the patient had a number of spontaneous partial remissions and subsequent relapses. Two days prior to death he suffered a sudden peripheral circulatory collapse from which he did not recover, despite administration of blood transfusions and adrenocortical extract.

Findings in Five Cases of Fatal Peripheral Circulatory Collapse

Case No.	Disease	Evidences of Infection		Adrenal Changes	Use of Cortisone or Corticotropin
		Clinically	At Autopsy		
1	Generalized exfoliative dermatitis	Skin	Flabby, friable, purplish spleen without malpighian corpuscles	Increased cytoplasmic granularity, decreased vacuolization	Yes
2	Generalized exfoliative dermatitis	Skin; bone marrow chronically hyperactive; splenomegaly	(No autopsy)	(No autopsy)	Yes
3	Generalized exfoliative dermatitis	Skin; bronchopneumonia	Toxic degeneration of heart and liver; bronchopneumonia	Twice normal size, with large circumscribed nodular mass in cortex	No
4	Generalized exfoliative dermatitis	Skin; bronchopneumonia	Bronchopneumonia	Thick bright yellow cortex; fascicular fatty hypertrophy	No
5	Lymphatic leukemia	Chill and temperature spikes to 104 F., blood culture positive (gram-positive coliform rods)	Acute bacterial endocarditis, lobar pneumonia, liver abscess	Hypertrophy of the adrenal glands (due to corticotropin)	Yes

At autopsy, the adrenals were found to be twice the normal size, with a large, yellowish, circumscribed nodular mass in the cortex. There was also bronchopneumonia, cloudy swelling of the heart muscle and of the liver parenchyma, and cortical cysts in both kidneys.

Case 4.—A 50-year-old white man suffered from generalized exfoliative dermatitis due to phenobarbital. This patient also died in shock, and at autopsy the adrenals showed a hypertrophic, bright yellow cortex. Microscopically, large amounts of lipids were present in the deeper layers of the cortex (zona fasciculata). Bronchopneumonia was also present.

COMMENT

The four cases reported here seem to us significant because all four presented certain identical or similar characteristics, which, in our opinion, were responsible for the patients' deaths in irreversible peripheral circulatory collapse. In the table an attempt is made to define the common pattern of these cases. The table also contains a fifth case from the literature,[4] that seemed to us to fit the pattern of the other four cases in regard to cause of death, although this patient did not have a generalized exfoliative dermatitis. This patient, a 67-year-old white man with lymphatic leukemia, had numerous nodules and tumorous infiltrations of the skin, lymph nodes, and inner organs, including the adrenals. He died suddenly

4. Sulzberger, M. B., and others: Effects of ACTH and Cortisone on Certain Diseases and Physiologic Functions of the Skin: I. Effects of ACTH. J. Invest. Derm. 16: 323-337 (May) 1951.

with a picture of acute infection: shaking chill, temperature rise to 104 F (40 C), gram-positive coliform rods in a blood culture, and peripheral vascular failure. This picture developed while the patient was in clinical remission apparently induced by repeated use of corticotropin, administered in a dose of 25 mg. four times daily. Autopsy showed leukemic infiltrations in the skin, acute bacterial endocarditis due to gram-positive cocci, lobar pneumonia, liver abscess, hypertrophy of adrenal glands (due to corticotropin administration), adenoma of the right kidney, and cortical cysts in both kidneys.

From the table it can be seen that in two of the cases (cases 3 and 4) the dermatitis was definitely due to a drug (due to phenobarbital in case 4 and due to unknown injections in case 3). A third case (case 2) developed possibly also after use of a drug (penicillin). The fact that three of the five cases were of drug origin and that these patients died in shock is in accordance with observations of others that shock is the cause of death in exfoliative dermatitis medicamentosa.

In all five cases there was evidence of multiple infection. The skin of the patients with exfoliative dermatitis showed extensive secondary pyogenic lesions. Bronchopneumonia, lobar pneumonia, endocarditis, liver abscess, or septicemia were present in four of the five patients (cases 2, 3, 4, and 5). The spleen and the bone marrow revealed changes characteristic of infection in two patients (cases 1 and 2). Toxic degeneration of parenchymatous organs due to infection was present in another case (case 3).

Fine [5] has recently shown in animal experiments that infection facilitated by and superimposed on tissue damage is responsible for the irreversibility of hemorrhagic shock. It seems reasonable to assume that the usually overwhelming infection in the cases reported here was similarly decisive in making the circulatory collapse irreversible, despite the use of high doses of antibiotics and despite the fact that these cases were not of the hemorrhagic type.

If we assume, therefore, that the infection combined with the extensive tissue damage was responsible for the resistance of the patients to the intensive treatment of the shock, there still remains the question of the primary cause of the circulatory collapse. In this respect an answer seems provided by the changes in the adrenal cortex in the four cases in which an autopsy was performed. In these, the adrenals were hypertrophic, as much as twice normal size, and on microscopic examination of sections taken from three patients (cases 1, 3, and 4) abnormal changes were seen in the cortices that appeared characteristic of degenerative and necrobiotic processes. The relationship between adrenocortical function and peripheral circulation is too well known to need any special discussion in this paper. It should merely be pointed out that according to Selye's concept [6] prolonged severe stress will produce exhaustion of the adrenal cortex. Pathological changes in the adrenal cortices, similar to those seen in our cases, have been observed in experimental animals subjected to severe stress.[7] Severe stress over prolonged periods of time was of course present in all our patients.

In summary it may therefore be said that the circulatory collapse in our patients was due to adrenocortical exhaustion as evidenced by the pathological changes found in these organs on autopsy. The extensive infections of the skin and of visceral organs, manifested by localized foci and by toxic changes of parenchymatous organs, were probably responsible for the irreversibility of the shock.

If this interpretation of our cases is accepted, then the occurrence of irreversible shock in generalized exfoliative dermatitis must be regarded as due to the same shock mechanism as the delayed shock deaths in extensive burns. Considering the clinical, physiological, and pathological similarity of exfoliative dermatitis and fatal burns, the identity of the shock mechanism in both of these conditions is certainly not surprising. On the contrary, it seems rather strange that no shock deaths have been reported in pemphigus, a disease that also shows clinical similarity to severe burns and exfoliative dermatitis, and in which, moreover, pathological changes of the adrenal cortex have also been found in a number of cases.

One more point concerning our cases is of great importance. Two of our patients (cases 1 and 2) and the patient in case 5 received treatment with cortisone and corticotropin. In view of reports on adrenocortical failure following substitution therapy or stimulation therapy with these hormones [8] the question may be raised whether the adrenocortical changes and consequently the shock deaths in these cases might not have been due to the use of these drugs. The lack of response to the second course of cortisone in the patient in case 1 and the failure of the eosinophil count to decrease in the Thorn test in this patient could well be interpreted as evidence of adrenocortical exhaustion. In the second case, the circulatory collapse occurred about three hours after resumption of corticotropin therapy with a 25 mg. dose, and an acute exhaustion of the adrenal cortex due to overstimulation might have occurred in this case. As much as these arguments tend to suggest a possible significance of the use of the hormones in regard to the development of the adrenocortical exhaustion, the occurrence of shock in the patients in cases 3 and 4, who had not been treated with cortisone or corticotropin, makes it clear that hormonal overstimulation is certainly not a necessary factor in the development of circulatory collapse in generalized exfoliative dermatitis. On the other hand, it cannot be denied that the hormones might have played a contributory role in the establishment of the shock; the autopsy report in case 5 describes even the adrenal hypertrophy explicitly as due to administration of corticotropin. On the whole, however, it seems more likely to us that any damaging overstimulation by the hormones was only an additional, and, at the worst, a precipitating factor rather than the main cause of the circulatory failure, since there can be no doubt that the diseases per se produced the major stress effect.

5. Fine, J., and others: The Bacterial Factor in Traumatic Shock, Ann. New York Acad. Sci. 55: 429-445 (Sept. 3) 1952.

6. Selye, H.: The Physiology and Pathology of Exposure to Stress: A Treatise Based on the Concepts of the General-Adaptation-Syndrome and the Diseases of Adaptation, Montreal, Canada, Acta, Inc., Publishers, 1950.

7. Selye, H., and Stone, H.: On the Experimental Morphology of the Adrenal Cortex, Publication 74, American Lecture Series, monograph in American Lectures in Endocrinology, edited by W. O. Thompson, Springfield, Ill., Charles C Thomas, Publisher, 1950.

8. Proctor, E. L., and Rawson, A. J.: Adrenal Cortical Failure Following Treatment with Cortisone and ACTH, Am. J. Clin. Path. 21: 158-162 (Feb.) 1951.

SUMMARY AND CONCLUSIONS

Irreversible peripheral circulatory collapse occurred in four patients with generalized exfoliative dermatitis of different origin (drug dermatitis, contact-type allergy, and lymphoblastoma). Degenerative changes in the adrenal cortex were found in three of the four cases in which autopsies were performed, and the adrenocortical failure manifested by these changes is believed to be responsible for the peripheral circulatory failure. Extensive infections of the skin and of inner organs were probably important factors in the therapy-resistance of the cases and the consequent irreversibility of the shock. Repeated use of cortisone and corticotropin in two of the four cases could also have been responsible for the occurrence of the circulatory collapse, by inducing failure of the adrenal cortex.

The relatively frequent incidence of shock deaths in drug dermatitis makes careful observation of these cases particularly important. Prevention of infection and constant control of peripheral circulation are essential in all cases of generalized dermatitis. The use of cortisone and corticotropin, although undoubtedly beneficial in most cases of generalized exfoliative dermatitis, should be strictly controlled and the dosage kept as low as possible. A second course of treatment should probably be avoided, or, if deemed absolutely necessary, the drug should be given in small dosage and only if the patient is in good general condition.

SCIENTIFIC EXHIBITS

Tom Jones
and
Thomas G. Hull, Ph.D., Chicago

It is obvious to all who attend medical conventions that the scientific exhibit is growing up. It is getting easier to look at and more effective year by year. Its improved quality and the respect that it now commands is the result of wider appreciation on the part of exhibitors of the simple but important rules of exhibit design. They are learning how to present their subject in a manner that will be both interesting and readily understood. They are learning also that the exhibit, like all other mediums for communicating knowledge, has certain peculiar and inherent capacities and limitations and that to be successful it must be designed to take advantage of the one and not exceed the other.

The purpose of this paper is to discuss some of the principles involved in effective exhibit production. It is written in the hope that exhibitors, particularly the new and less experienced ones, may avoid the errors and practices that even today make dull and ineffective many an otherwise excellent exhibit story. We have carefully avoided mention of formulas or specific procedures for fear that they would lead to a stereotyped approach or pattern of exhibit design. This would, in our opinion, be fatal, since the continued improvement of the exhibit medium depends to a large extent on the exhibitors' own judgment, originality, and good taste.

Within its limitations of time and space, the exhibit is probably the best device that man has invented to communicate facts and ideas about the physical world. The reason for this is that the exhibit combines the advantages of visual mediums and language into a specific coordinated technique that has a higher teaching potential than any other known modality.

In the field of medicine the scientific type of exhibit has come to be regarded as a practical and highly efficient instrument for communication of knowledge and ideas. The growth of their popularity has been so rapid in recent years that they are today the chief attraction at many of the larger annual conventions. Here the exhibit's inherent capacity to convey information and broaden understanding is further increased by the addition of the personal element. This is provided by the presence of exhibitors who can demonstrate, discuss, and answer questions while surrounded by the visual materials of their respective subjects. While the conventions are in progress, the exhibit booths become the common meeting ground of physician, scientist, teacher, and student. Here they may see graphically presented the newest developments in clinical and research medicine and through study of the displays and exchange of ideas may keep abreast of medical progress, practices, and trends.

WHEN TO USE AN EXHIBIT

When should a scientific exhibit be the medium of choice in reporting the results of investigation? The answer depends mainly on the subject. Not all subjects lend themselves to presentation by exhibit. Some can best be communicated by a lecture (with or without slides). Others, involving movement and dynamic processes, can only be shown effectively by the motion picture. The exhibit medium is applicable only when the subject can be put into visual form, with photographs, drawings, models, graphs, specimens, or the actual demonstration of a procedure. The visual impression that a good exhibit makes on the visitor is a lasting one and is well worth the effort and expense of preparation, but not all subjects lend themselves to the exhibit technique. The "textbook" type of exhibit, with placards containing one paragraph after another of text material and no illustrations, is not a good exhibit. In fact it is not an exhibit at all but an enlargement of something that belongs on a printed page. There is no visual impression from such a display, nothing to arouse the observer's interest, and nothing that he is likely to look at or afterwards remember. Bright lights, pleasing colors, and beautiful woodwork are useful adjuncts when properly employed, but that is all they are. They do not make an exhibit; they tell no story nor convey any message.

Professor of Medical and Dental Illustration, University of Illinois (Mr. Jones); Director of Scientific Exhibit, American Medical Association (Dr. Hull).

The decision to have an exhibit carries with it certain obligations. The acceptance of an exhibitor's application for space at a medical convention assumes that he has something worthwhile to say. It also assumes that the material he will present has been well considered, that statements made will be sound and valid, and that credit will be given to others where it is due. It is inevitable that the exhibit story will, to some degree, reflect credit or discredit on the institution that the exhibitor represents. It is, therefore, important that proposals for exhibits by staff members of an institution be carefully scrutinized before endorsement is given. An effective method of handling this is through an exhibit committee that passes on all exhibit proposals.

Along with their virtues as mediums of communication, scientific exhibits have certain inherent disadvantages that should be kept in mind. They can function at one time and in one place only, and unlike printed literature and pictures that can range far and wide and be studied at will, they are immobilized in one spot and therefore limited to those who can seek them out within the time they are on display. Herein lies the basic reason why the exhibit must be a highly functional unit. It must tell its story swiftly and with precision.

THE COST FACTOR

An ever present factor affecting the employment of scientific exhibits is cost. While this is not excessive, it is a practical factor to be taken into consideration. A worthy exhibit, intended for use at the larger medical conventions and prepared according to modern standards, requires time and almost always the special skills of an artist or experienced technician. It must then be transported to a distant point and installed. Here it must (or should) be attended throughout the meeting by an exhibitor or associate who can demonstrate it and answer questions. The total expense involved naturally puts a strain on the budgets of some institutions and individuals who might otherwise make wider use of the exhibit medium.

The cost of a scientific exhibit can properly be calculated only on the basis of its ultimate investment value to the exhibitor and to medical education. This in turn depends on the total number of persons (and the kind of persons) who will see the exhibit in proportion to its total cost. Other considerations sometimes exist, but the principle of cost per consumer remains. It is the same principle that determines the cost of radio programs and advertising projects and can be expressed as follows:

$$\text{Index of Investment value} = \text{Number of Observers} \div \text{Cost of Exhibit}$$

In other words, an exhibit costing $100 that is seen by only 100 persons is 10 times as expensive as one costing $1,000 and seen by 10,000 persons.

The total number of persons who see a particular medical exhibit depends, of course, on the number of occasions on which it is shown. For instance, if it is originally displayed at an American Medical Association convention and later shown at a sectional or state meeting, followed by one or more local meetings, the total may reach a high figure. This is further increased when the exhibit can be set up in a teaching institution after its travels are over and exposed to students and staff for a more extended period. Generally speaking, the life span of a scientific exhibit in medicine averages about three showings. This varies considerably, however, according to the timeliness and popularity of the subject and how interestingly it is presented.

The exhibitor assumes a responsibility to the observer when he undertakes a scientific teaching exhibit that many do not seem to realize. Not only must the facts and data that he offers be scrupulously accurate and his conclusions sound and free from any statements that may be misleading, but his exhibit should have unity, clarity, and interest. Unless it is designed to be comprehended without undue tax on the observer's time, it will fail in its objective. The time factor is an important consideration. "Comprehension time" is a convenient term to express the number of minutes required by the average observer to grasp the meaning and significance of a particular exhibit. Obviously this will vary with the individual observer's interest, experience, mental acuity, and visual mindedness. It is a valuable yardstick, however, to measure the relative effectiveness of the exhibitor's efforts under different conditions. One application is shown in figure 1.

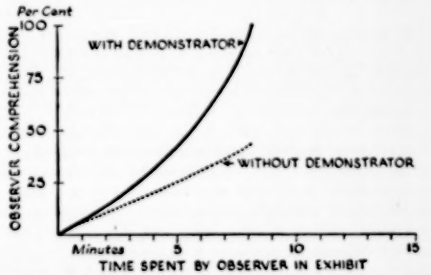

Fig. 1.—Hypothetical graph based upon study of observer reaction. The high value of the demonstrator will vary with exhibits, but invariably the presence of a demonstrator will greatly enhance the over-all effectiveness of the exhibit.

A good exhibit, carefully prepared, attracts the observer and invites study. By the same token a bad one repels. It is well to remember that, while an exhibit can reflect credit on the exhibitor, it can also work the other way. If it is in poor taste or cluttered, confused, and overcrowded, few will bother to look at it. Those who do will carry away an opinion that is anything but flattering to the exhibitor.

PLANNING THE EXHIBIT

If the scientific exhibit is to be worthy of its name, it must be thought out and planned beforehand. If it is to function and tell its story, it must have calculated order and design. This is step number one—the most important of all—and all else follows it, including success or failure. A carefully worked out plan will save time and money and, other things being equal, will do more to insure the success of the exhibitor's efforts than any other factor.

One frequently hears the question, "How do you go about planning an exhibit?" Actually there is nothing very difficult about it although, like any creative effort such as writing a paper or painting a mural, one has to

first learn the basic principles involved and how to apply them to the task. These principles are (1) unity (2) clarity (3) accuracy, and (4) brevity. It will be observed that these are the requisites for all forms of communication of information regardless of the medium employed. In none, however, are they more important than in scien-

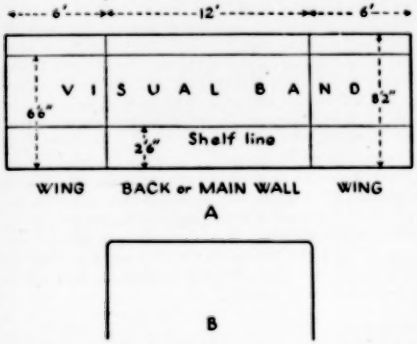

Fig. 2.—*A*, plan of 12 ft. exhibit space. Such a plan made to scale, 1 (or 2) in. to the foot, drawn on strong paper is the first step in the planning of a scientific exhibit. It enables the exhibitor to organize material beforehand and determine proper size and placement of mediums used. *B*, shows how plan may be bent to show shape and floor space of booth. It is often advisable to make a cutout plan on cardboard and construct booth in miniature.

tific exhibits. Here the story or message must be given in its entirety within a short space of time. The observer cannot refer back to it as he can in a written article, nor is it likely to be available to him ever again except for the one time he sees it. Another reason why careful planning is necessary is that an exhibit cannot command the exclusive time of the observer, but is usually shown side by side with many others, all competing for attention. It may be safely assumed that the observer, with so many things to see and so many demands on his

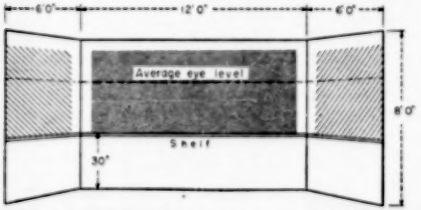

Fig. 3.—Diagram showing usable exhibit area. The relative importance of the space is indicated by degrees of shading. Note that the back wall takes precedence over the wings.

time, is not likely to linger at an exhibit that looks involved or one that demands much effort on his part to comprehend.

The first step in the design of a scientific exhibit is to make a plan on paper. This should be done to scale using some such convenient ratio as 1 in. to a foot (some prefer the scale of 2 in. to the foot). First, the back wall of the exhibit space should be outlined, which is the number of feet assigned. Then, at each side, the wing space should be added, which usually exists although it is not included in the number of feet assigned to the exhibitor. If the wings are drawn at a slight angle on the plan, it will help in visualization of their relationship to the back wall (fig. 2).

Then a line should be drawn across the plan at a point 2½ ft. from the floor line. This is the shelf level (if one is provided) or lower limits of the space that may be used for exhibit material. A corresponding line should then be drawn 6½ ft. from the floor to mark the top limits of the usable space. This gives a band 4 ft. high extending across the entire space including wings. This is the visual band or usable area above or below

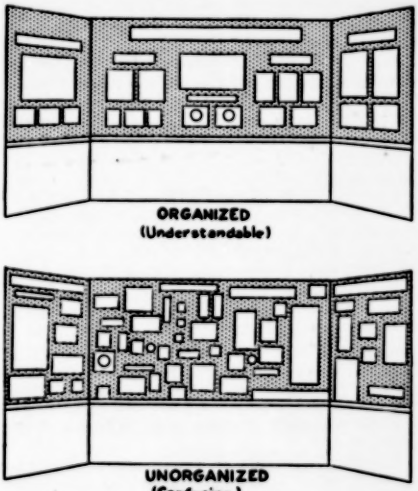

Fig. 4.—Portrayal of the difference between a well-composed exhibit and the confused or helter-skelter type.

which the display material will not be clearly seen. Of course, titles and headings may be placed above this top line.

Before indicating on the plan the various items of the exhibit and their arrangement, it is well to bear in mind that in most cases the back or main wall must tell the exhibitor's story. The wing areas are useful also but should be regarded as secondary in importance to the back wall. The space of some 6 to 8 in. to either side of the point of junction of back and side wall should not be used if possible, since wall material within this area cannot be viewed at a comfortable angle (fig. 3).

In a typical 12 ft. booth the usable wall area will total about 85 sq. ft. Although this is something less than half of the gross space, it is all that the observer can see naturally and without strain. In figure 3 the relative importance of this exhibit area is indicated. This is based upon the eye habits of the observer and

is a constant which, if kept in mind, should guide and simplify the work of the designer.

It is usually a good idea to consider the center of the back wall as the focus or center of attraction. For this reason it is often wise to have at this point a brief statement of the purpose and point of the exhibit. A study of eye habits of exhibit observers would seem to show that the eye wanders to right and left of center, is arrested by striking or pertinent material and ends its journey at the wings or point farthest from center.

With the exhibit space clearly visualized by the scale plan, and the fixed physical factors in mind, one can proceed to work out in pencil on the plan a logical arrangement of the material of the exhibit. Procedure from here on will depend upon the nature and purpose of the exhibit, what material is available, etc. (fig. 4).

There are several types of scientific exhibits, ranging all the way from demonstration on a living human model to a display of pure statistics. The first utilizes chiefly the floor space of the exhibit area, the second chiefly the wall. The typical scientific exhibit with which we are concerned here however will be composed of flat pictorial or lettered wall material plus (in many cases) specimens, models, or other three dimensional objects. The material used will, of course, be dictated by its appropriateness to the job of communicating the exhibitor's ideas clearly and effectively. To do this, the exhibit must above all be graphic. Descriptive text should be kept at a minimum, and statistical tables, graphs, legends, and all word material should be simple and clearly and tersely expressed. The temptation to add more material than necessary to tell the story should be resisted. Nothing detracts from the appearance and effectiveness of a scientific exhibit so much as crowding the space with more material than can be readily assimilated.

If the exhibit proper has presented the main or the salient points of the story in a graphic and readily understood manner, it has fulfilled its mission. All the related facts, figures, and details that the exhibitor feels are essential to the whole story may be mimeographed or printed and passed out to all who wish to have such information. This has the double advantage of giving the visitor material for reference and further study while at the same time permitting the exhibit to be designed in a proper and interesting manner. Experience has shown that this practice is far more effective than an overloaded and crowded exhibit, and furthermore it widens the scope of the exhibit story while earning the gratitude of the weary visitor.

An important factor in the success of a scientific exhibit is lighting. No matter how carefully it has been designed, the exhibit's effectiveness is sharply reduced unless it is well lighted. Ordinarily the general lighting of the hall or room where exhibits are shown is insufficient to give proper visibility to the displays. Accessory lighting in the form of brackets placed along the top of exhibits is provided by some of the larger organizations such as the American Medical Association. If it is not provided, however, arrangements can usually be made to have it supplied. The scientific exhibitor can profit by the experience of shopkeepers, who have long ago learned that nothing contributes to the appeal of their displays so much as good illumination.

Following is a list of suggested principles to be followed in preparing an exhibit:

1. The exhibit should be planned well ahead of deadlines, and preparations started early.
2. The main theme of exhibit should be clear at a glance.
3. All material should contribute directly to the story.
4. The size and style of lettering is important; typed copy is too small unless enlarged.
5. Assigned exhibit space is important; it should be used economically.
6. The floor space should not be cluttered with furniture. It prevents access to exhibit.
7. Over-elaboration, too bright colors, and startling effects should be avoided. Such things are distracting.
8. Transparencies are effective, but expense is a factor in their use.
9. Transparencies 2 by 2 in. in size are too small to be used.
10. Material should be placed in easy eye range.
11. Graphs should be simple and bold; pie and bar graphs preferred.
12. No glass or cellophane should be used over pictures; both cause glare.
13. If unattended, an exhibit can lose as much as 50% of its value.
14. The exhibit should be assembled before it is sent to the meeting and checked carefully.
15. A good exhibit is not necessarily costly to produce. Skill and good taste are more important than money.

It is hoped that the foregoing observations will answer some of the questions that so often arise in connection with scientific exhibit production. There can be no question that the exhibit medium has become an important channel for the presentation of new ideas and developments in medicine. As such it is worthy of our best efforts.

Therapy of Congestive Heart Failure.—It has become generally acknowledged that the reduction of sodium chloride to 2 gm. per 24 hours is inadequate in many patients [with congestive heart failure] and that diets containing 200 mg. or less of sodium are practical. The old Karrell diet of 200 cc. of milk four or five times per day contains about 800 to 1,000 mg. of sodium chloride and is not the best diet for the severe forms of congestive failure over prolonged periods of time.

In the years of fluid restriction without salt restriction, patients were miserable from thirst. It is now recognized that with proper restriction of salt the patient may be permitted enough water to ensure comfort. Although some have advocated forcing fluids to levels as high as 5,000 and 6,000 cc. per day, the value of such a regimen is doubtful.

The modern regimen of salt restriction and mercurial diuretics has resulted in improved management of congestive heart failure, lessened disability and greater longevity. But the use of a powerful tool such as marked sodium restriction also has introduced dangers. Sodium depletion and other electrolyte imbalance, particularly in patients with renal disease who are placed on a rigidly restricted sodium intake, has led to the development of weakness, anuria, and azotemia, which, when unrecognized, has resulted in fatalities. The use of mercurial diuretic agents in such patients has heightened the incidence of such reactions. In other patients with renal impairment, mercurial diuresis has resulted in a disproportionate loss of chloride with hypochloremic alkalosis. These untoward complications may, however, be avoided by awareness of their occurrence, early diagnosis and immediate correction of the electrolyte imbalance.—H. L. Blumgart, M.D., The Management of Congestive Heart Failure, *Circulation*, January, 1953.

EXPERIMENTATION IN MEDICAL EDUCATION

H. G. Weiskotten, M.D., Skaneateles, N. Y.

When the Council on Medical Education was established by the American Medical Association in 1904, its function was clearly stated to be the improvement of medical education. That the American Medical Association looked to medical educators to assume leadership in the improvement of medical education is indicated by the membership of the first Council, which was as follows: Arthur D. Bevan, M.D., professor of surgery, Rush Medical College, chairman; W. T. Councilman, M.D., professor of pathology, Medical School of Harvard University; Charles H. Frazier, M.D., professor of surgery, University of Pennsylvania School of Medicine; Victor C. Vaughan, M.D., dean, University of Michigan School of Medicine; and J. A. Witherspoon, M.D., professor of medicine, Vanderbilt University School of Medicine.

Immediately after its establishment, the Council initiated the Annual Congress on Medical Education. These congresses have been occasions for reviewing the progress being made in the improvement of medical education. They have provided opportunities for full and free discussion of current problems as well as reports on newer methods of teaching.

EARLY PROBLEMS

Two major problems faced the leaders in medical education during those early years. It had become obvious that a large number of inferior medical schools that offered no promise of satisfactory development should be eliminated. It was equally apparent that if the educational and scientific life of medical schools was to flourish, it would be highly desirable for them, wherever possible, to develop university affiliations and to foster the development of university atmosphere and standards.

One of the first actions taken by the Council to meet these problems was the establishment of a set of educational essentials that schools had to meet in order to be included in the Council's list of approved medical schools. These early essentials contained rather rigid minimum requirements for faculty personnel, curriculum, and physical facilities. They even went so far as to specify the number of hours to be devoted to the various subjects to be included in the medical curriculum. Similarly rigid standards were adopted by the Association of American Medical Colleges. Although the educational hazards inherent in the establishment of such rigid requirements were recognized at the time they were formulated, they were deemed temporarily necessary as a means of eliminating a large number of hopelessly inferior proprietary medical schools then flourishing throughout the country.

The application of these early standards, together with the impact of the Flexner Report, and the cooperation of most of the state boards of medical licensure were successful in eliminating most of the substandard schools. A few with limited regional recognition continued to function, however, until very recently. Today we can point with pride to the fact that there is not a single unapproved medical school operating in the United States.

The signal success of these initial accreditation programs did not render them immune to criticism. Medical education in this country was accused of having become routine and mechanical. This criticism is still made both in this country and abroad by those who have not followed closely the subsequent development of our medical schools. The charge is sometimes made that the accreditation programs have stultified rather than encouraged educational experimentation and improvement in medical education.

Actually nothing could be farther from the truth. As soon as it was considered safe to do so, the detailed rigidity of all requirements was eliminated and schools were encouraged to develop their own individual programs as long as they provided an over-all satisfactory program of undergraduate medical education.

At the same time advancements in the field of medicine as well as advancement in educational methods have placed on the schools ever increasing responsibilities in their efforts to provide their students with a sound foundation for careers in medicine.

In the early years the major problem of the schools was to develop basic science departments staffed with competent, fulltime scientists devoted to teaching and to the development of new knowledge in their special fields. Well-equipped laboratories had to be provided not only for undergraduate teaching but also for graduate training in these fields and for the research of the teaching staffs.

Efforts were thus made to apply to the undergraduate medical course the sound fundamental principle of education that the best education is self-education and that the responsibility of a medical school is to provide the facilities, stimulus, and guidance for students to educate themselves. The close supervision necessary to the successful operation of such programs required great increase in the number of fulltime teachers, thus adding to the problem of faculty recruitment and further increasing the budgetary requirements of the schools.

Nevertheless the successful application of these fundamental principles of education in the basic medical science departments led to experimental efforts to apply them in the teaching programs of the clinical departments. It was obvious that to implement such programs it would be necessary to revamp completely the curriculum of the clinical years. Their introduction required the use of the hospital wards and outpatient clinics as laboratories for the teaching of clinical medicine.

The schools were thus faced with the necessity of securing satisfactory clinical facilities that would be under their control. Most important of all, it was necessary to secure a sufficient number of competent teachers who would devote the increased amounts of time required by this type of teaching as well as maintain high standards

This paper was read at the Annual Congress on Medical Education and Licensure, Feb. 9, 1953.

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of medical services in their various departments. Furthermore, such competent teachers interested in academic careers sought opportunities for research as well as teaching. These were the problems incident to the development of clinical clerkships.

As these programs were finally developed, emphasis was placed on the student acquiring an understanding of the fundamental principles of the basic medical sciences and their application in the field of clinical medicine. Emphasis was placed on attitudes, habits of thought and work, and the proper approach to problems in medicine rather than on memorizing from textbooks and lectures innumerable facts relating to all of the various phases of medicine. In other words efforts were made to provide for the student a training for the practice of scientific rather than empirical medicine.

These developments based on years of experimentation have tended to individualize medical education to an extent far beyond that attained by any other professional schools. The problems involved have been enormous and still remain to be satisfactorily solved by quite a number of our medical schools. Incidentally, they have required relatively enormous increases in the budgets of the schools but have assured the American public of standards of medical care not otherwise obtainable.

NEW PROBLEMS AND EXPERIMENTATION

Having developed such improved teaching methods in both the basic medical science and clinical departments, the schools were faced with new and complicated problems. Perhaps the most fundamental educational problem was how to integrate the programs of the various departments to best qualify a student to initiate his career in medicine. Solutions to this problem have required a meeting of the minds of the teachers in the various departments of a school and the recognition of common broad objectives of the undergraduate curriculum as an integrated whole. They have been the basis for much experimentation by individual schools. There is undoubtedly no single solution that would be applicable to all of the schools. It is the responsibility of the faculty of each school to continually reevaluate its curriculum and to provide in accordance with its own particular setting a sound and well integrated educational program. In this connection may I repeat a statement that I made before this congress two years ago. No successful educational program can be dictated by our Council, by any outside agency, or even by the administration of a medical school. It can be no better than the competence and interest of those directly responsible for its conduct.

Each year this program of integration of the undergraduate program becomes more complicated and more difficult. Continuing advances in the various fields of medicine present problems of expanding the content of the existing overcrowded curriculums of the schools and of adding new subject matter.

Frequently such new subject matter has not appeared to fit readily into the programs of existing departments, and attempts have been made to present it to the students in didactic form as separate courses detached and isolated from the preexisting curriculum content. Such attempts have not been too successful, and the schools have been faced with the problem of attempting to incorporate such new subject material into the individual experiences of the student in the integrated programs of the basic medical science and clinical departments.

Here again the solutions of these problems have required not only continued reevaluation of the course content of the various departments but also continued experimentation as to how in a given school important new subject matter and viewpoints can be best integrated into the basic training of the student.

This seems to be especially true when the new subject matter is considered to be outside the fields of the basic medical sciences. For example, for years now there has been a growing appreciation that there are a host of psychological, sociological, economic, and environmental factors that must be taken into consideration and dealt with by a physician if he is to meet his responsibilities in the prevention, diagnosis, and treatment of disease in his patients. Just how to prepare a student to appreciate these factors and to be prepared to deal with them has presented a real problem and has given rise to considerable experimentation for more than 20 years. The fact that the development of our educational programs has been based largely on the natural sciences and that we have required some preliminary training in these sciences for admission to our medical schools adds to the difficulty of incorporating these newer concepts into the undergraduate curriculum. Here again, as has been the case in training students in the natural sciences, the question arises as to how much of the social sciences we should attempt to include in the training of the undergraduate medical student and further how it can be best integrated into his individual experiences. Certainly we should find some way of developing in the student proper viewpoints and attitudes toward these problems. At the same time we must accomplish this without any interference with a sound training of the student in medicine as it has been developed on the basis of the natural sciences. There are many who believe that just as a student is required to have some preliminary training in the natural sciences before entering upon the study of medicine he should also have similar preliminary training in the social sciences if he is expected to apply them in his medical training.

Other experiments are being attempted in an effort to influence the nature of practice students will adopt for their future careers in medicine. For example, some schools have attempted to incorporate in their undergraduate curriculums influences and experiences that will result in a larger percentage of their graduates entering the field of general medicine rather than limiting their practice to a specialty.

The recognition of all of these and other problems by the medical schools together with the experiments that are being undertaken to find solutions of them offer great promise for the continued improvement of medical education. Although for a period the effect of the depression and the war on the medical schools tended to retard or block extensive experimentation, there are many signs that we have entered a more fruitful era of experimentation if medical education is not again dislocated by another emergency.

At the present time many experiments are currently in progress. To cite a few of the schools that are con-

ducting experiments in various important phases of medical education mention may be made of Boston University, Bowman-Gray School of Medicine of Wake Forest College, University of Colorado, Cornell University, University of Pennsylvania, University of Washington, and Western Reserve University.

While experimentation is to be welcomed and encouraged, it should be pointed out that in undertaking experiments schools should be cautious that they lose none of the progress that has been made in placing medical education on a sound educational, scientific, and individual basis. Furthermore it should be realized that it takes time to develop an intelligently designed experiment. To develop a successful experiment a school must first analyze its objectives and resources. If this is done, almost any experiment based on sound educational principles and carried out by a competent and enthusiastic faculty offers promise of serving as a contribution to the improvement of medical education.

It should be kept in mind, however, that it may be extremely difficult to evaluate the results of experiments in medical education. Such evaluation may require continuing observation of their influence on students for several years after graduation.

Finally, just as individual faculty members are continually conducting research for the advancement of medical science in their particular fields, as faculty groups they should continually be conducting experiments in the field of medical education—for this is the primary responsibility of our medical schools and must always be recognized as such.

Postoperative Care.—Of all the numerous factors which contribute to a successful outcome following a surgical procedure, there is none more important than careful observation. Intelligent postoperative care does not lend itself to remote control, and someone familiar with the great variety of signs and symptoms diagnostic of changes within the surgical patient's economy must pay attention and interpret these meaningful messages in the light of possible prophylactic or therapeutic action. The sudden pallor, restlessness, air hunger, and rapid pulse, indicative of massive internal hemorrhage, are bits of evidence noted and interpreted by observation at the bedside.

There is a growing tendency on the part of surgeons to forego the correlation of laboratory tests with bedside patient evaluation and direct the postoperative care merely by the recordings in the laboratory report forms. Obviously such a policy exposes the physician to numerous dangerous therapeutic pitfalls and results in inadequate and erroneous observation of complications.

Of significance to the patient's general comfort and also to the ultimate recovery are many minor items, any of which alone might not seem important but in the aggregate contribute substantially to the patient's welfare and eventual recovery. Careful, intelligent, and constant observation alone can detect such signs. The examining finger, for example, is of greater value than paregoric in the diagnosis and therapy of a diarrhea resulting from a rectal fecal impaction. Subumbilical pain and discomfort accompanied by a suprapubic fullness following a spinal anesthetic or abdominal operative procedure may be relieved in most cases much more effectively by a catheter than by sedation. Intelligent laboratory tests can be carried out only by close patient-physician relationship as interpreted by frequent and careful inspection, palpation and auscultation.—M. A. Casberg, M.D., Postoperative Care, *Surgery*, February, 1953.

CLINICAL NOTES

PANCYTOPENIA FOLLOWING USE OF HYDRALLAZINE ("APRESOLINE")

REPORT OF A CASE

Manuel Kaufman, M.D., Brookline, Mass.

The purpose of this paper is to discuss a potential hazard from the use of hydrallazine ("apresoline"), which is being given currently for treatment of hypertension. Favorable response has been reported with this drug in various types of hypertension, and some excellent results have followed its use in persons who have failed to benefit from sympathectomy.[1] It is said to lower blood pressure through central action, perhaps at the level of the hypothalamus, and has a partial adrenergic blocking effect. Supposedly, it does not affect the myocardium adversely or decrease the renal blood flow.[2]

REPORT OF A CASE

A woman, aged 44, has been a known hypertensive for 20 years. Her hypertension developed following bilateral chronic nephritis first acquired during pregnancy. Her systolic blood pressure at the time was 180 mm. Hg. She manifested symptoms of headache and cardiac embarrassment with associated grade 3 changes in the ocular fundi (Keith, Wagener, and Barker [3]). A bilateral lumbodorsal sympathectomy was done six years ago, with the development of moderate postural hypotension following the second stage. Benefits from the operation were obtained for one year, and then the blood pressure rose to levels as high as 260/140. Medical therapy was instituted and included administration of sedative drugs and restriction of sodium chloride intake to 400 mg. a day. The patient gradually resumed her normal activities as a housewife; periodic courses of antibiotics were administered to control urinary tract infection. She manifested sensitivity to the sulfonamides by a skin eruption but had no depression of peripheral blood elements. Two years ago, because she had recurrence of headache, dyspnea with slight exertion, and rise in blood pressure, "veriloid," a derivative of veratrum veride, was administered in a dosage just below the emetic range. Her blood pressure was lowered to 140/110 in the right arm and 140/90 in the left arm, but as this medication was continued there was gradual development of resistance to the beneficial effects of the drug.

In early December, 1951, the patient complained of dyspnea on slight exertion, palpitation, headache, and fatigue. Her blood pressure was 260/150. On hospital admission, funduscopic examination revealed increased light reflex with a venous arteriole ratio of 3 to 1. Small linear hemorrhages were observed. The left border of the heart was prominent, and a forceful apical impulse was palpated. A grade 2 systolic murmur was heard at the mitral area, and the aortic second sound was greater than the pulmonic second sound. The lung fields were clear. The electrocardiogram showed the presence of left ventricular strain. Intravenous pyelographic examination showed delayed appearance but good concentration of the dye

1. Schroeder, H. A.: The Effect of L-Hydrazinophthalazine in Hypertension, Circulation **5**:28-37, 1952.

2. Craver, B. N., and Yonkman, F. F.: Some Pharmacological Properties of L-Hydrazinophthalazine, a Hypotensive Agent, Federation Proc. **9**:265-266, 1950.

3. Keith, N. M.; Wagener, H. P., and Barker, N. W.: Some Different Types of Essential Hypertension: Their Course and Prognosis, Am. J. M. Sc. **197**:332-343, 1939.

on the left. The right excretory system was not well visualized, and calices of the left were blunted and rotated. There was delayed excretion into the bladder. The observations suggested renal infection and impairment of renal function. On Dec. 10, the white blood cell count was 4,550 per cubic millimeter, with a differential count of 67% polymorphonuclear cells, 27% lymphocytes, 3% monocytes, and 3% eosinophils; the hematocrit was 43.5%; the hemoglobin level was 15.1 gm. per 100 cc. (photoelectric colorimeter); the mean corpuscular hemoglobin concentration was 34.7; the thrombocytes appeared normal. On Dec. 22, the white blood cell count was 5,900. Urinalysis showed 2+ albumin, two to three white blood cells per high power field, and occasionally a hyaline cast. Results of a urine concentration test were as follows: specific gravity after one hour, 1.005; after two hours, 1.004; and after three hours, 1.003. Phenolsulfonphthalein sodium test results were 17% in 15 minutes and 50% in two hours; the fasting blood sugar level was 88 mg. per 100 cc.; the nonprotein nitrogen level was 50 mg. per 100 cc.; the serum chloride level was 100 mEq. per liter; and the icteric index was 5; a urine culture grew nonhemolytic Enterococcus and Staphylococcus albus.

During the patient's hospital stay of two and a half weeks, hydrallazine was given in a dose of 25 mg. four times a day. The patient experienced nasal stuffiness, occipital headaches, tachycardia, numbness, and tingling of the hands and legs during the first three days of therapy. The dose of hydrallazine was increased in increments of 25 mg. until the desired effect was obtained. For one month, combined hydrallazine and "veriloid," therapy was given, the dose of the latter being 2 mg. four times a day. The "veriloid" was subsequently omitted because of nausea. Seven weeks after therapy with hydrallazine, ecchymotic areas were observed over the dorsum of the hands and both hips. The blood pressure was 130/90 in the supine and erect position; the pulse rate was 80 beats per minute. The white blood cell count was 3,800 and the red blood cell count 3,300,000 per cubic millimeter. Hydrallazine therapy was stopped for two weeks, and the white blood cell count was rechecked and found to be 5,700; the platelet count was 240,000 per cubic millimeter (Rees and Ecker, normal 150,000 to 300,000). Hydrallazine therapy was again instituted in a dose of 25 mg. four times a day for seven weeks. Because of severe headaches and elevation of the blood pressure to 190/110, the dose was increased to 50 mg., 100 mg., 100 mg., and 50 mg. at four hour intervals. It was necessary, finally, to increase the dose to 100 mg. four times a day to control symptoms. On this dose schedule the patient complained of cramps in her legs, and ecchymotic areas were observed on the arms and legs. One month later, her bleeding time was two and one-half minutes; the white blood cell count was 5,600, with a differential count of 56% polymorphonuclear cells, 4% band cells, 38% lymphocytes, and 2% eosinophils. A capillary fragility test (Gothlin method) was done and two petechiae were seen. The patient described a sense of depression and a desire to cry while on the higher dosage of the drug.

Within four weeks, the patient was admitted to the hospital because of chills and fever of 24 hours duration. Physical examination revealed multiple ecchymotic areas scattered over the trunk and extremities, varying from a few millimeters to several centimeters in diameter. The temperature was 101 F, the pulse rate 100, and the blood pressure, 120/80. Funduscopic examination showed increased light reflex, and the venous arteriole ratio was 2 to 1. There was grade 1 sclerosis of the arterioles, and no fresh hemorrhage or exudate was seen. The ears, nose, and throat, and breasts were normal, and the thyroid was not palpable. The lung fields were clear. Bilateral curvilinear scars were present from the dorsal to the lumbar area (dorsolumbar sympathectomy). The left border of the heart was observed on percussion to be at the midclavicular line. There was normal sinus rhythm and a rate of 100 beats per minute, and a grade 2 apical systolic murmur was heard; the aortic second sound was accentuated. Tenderness was elicited in the left costovertebral area, and the lower pole of the spleen was palpable; this regressed after one week. There were no other palpable masses. Results of rectal and pelvic examinations were normal.

On July 14, 1952, the results of laboratory tests were as follows: hemoglobin level 9.76 gm. per 100 cc., hematocrit 32%, red blood cell count 3,320,000, white blood cell count 2,050, with 62% polymorphonuclear cells, 8% band cells, 28% lymphocytes, 0 monocytes, and 2% eosinophils, and platelet count 48,000; urinalysis showed a specific gravity of 1.010, pH 7.5, no sugar, albumin level 138 mg. per 100 cc., 15 to 20 white blood cells and no red blood cells per high power field, and no granular casts; culture regrowth from a catheterized urine specimen showed Escherichia coli sensitive to aureomycin, chloramphenicol, and oxytetracycline ("terramycin") and resistant to penicillin and streptomycin; serum sodium level 137 mEq. per liter, serum potassium level 4.4 mEq. per liter, and nonprotein nitrogen level 50 mg. per 100 cc. On July 16, results of blood studies were as follows: hemoglobin level 9.5 gm. per 100 cc., red blood cell count 2,600,000, white blood cell count 1,950, with 68% polymorphonuclear cells, 3% band cells, 13% lymphocytes, 1% eosinophils, and 15% large lymphocytes. On July 17, results of blood studies were as follows: hemoglobin level 11.1 gm. per 100 cc., white blood cell count 4,200, with 71% polymorphonuclear cells, 21% lymphocytes, 7% monocytes, and 1% eosinophils, and platelet count 74,000. On July 22, results of blood studies were as follows: hemoglobin level 12.1 gm. per 100 cc., red blood cell count 3,960,000, white blood cell count 3,400, with 79% polymorphonuclear cells, 3% band cells, and 18% lymphocytes, and platelet count 62,000.

A sternal bone marrow aspiration showed hypocellularity of elements with no total deficiency. Megakaryocytes appeared normal, and thrombocytes were slightly decreased. The erythroid-myeloid ratio was 3 to 1. Because of the pancytopenia, hydrallazine therapy was discontinued. A transfusion of 500 cc. of whole blood and oxytetracycline ("terramycin") by mouth, 500 mg. every six hours, were given. Within 48 hours there was a prompt drop in temperature, with clearing of pyuria. Oxytetracycline therapy was continued for one week. Four days after hydrallazine administration was discontinued, the blood pressure rose to 180/110. A preparation of Rauwolfia serpentina [4] was given, and the blood pressure dropped to 130/80. Within 10 days, the ecchymotic areas of the skin faded considerably, without further development of new lesions. Two and one-half weeks after the patient's discharge from the hospital, the white blood cell count was 4,000, with a normal differential count (58% polymorphonuclear cells, 4% band cells, 36% lymphocytes, and 2% eosinophils), and the platelet count was 130,000. The liver, kidney, and spleen were not palpable, and there was no regional adenopathy. The blood pressure was 120/90 in the right arm, and 130/80 in the left. Administration of ½ tablet of the R. serpentina preparation twice daily has been continued.

COMMENT

This patient manifested a pancytopenia after being treated with hydrallazine ("apresoline") for seven months. She showed signs of sensitivity to the drug during the early months of therapy. The toxicity of hydrallazine has been investigated by Meier and co-workers.[5] They did blood studies on dogs before administering hydrallazine and repeated them 45 days afterward, but noted no essential change in the blood picture. Schroeder reported that he had administered hydrallazine orally to patients for one to forty weeks without deleterious hemopoietic effects.[6] This patient's pancytopenia was reflected

4. Wilkins, R. W.: Personal communication to the author.

5. Gross, F.; Druey, J., and Meier, R.: Eine neue Gruppe blutdrucksenkender Substanzen von besonderem Wirkungscharacter, Experientia **6**: 19-21, 1950.

6. Schroeder, H. A.: Effects on Hypertension of Sulfhydryl and Hydrazine Compounds, J. Clin. Investigation **30**: 672-673, 1951.

mainly in the peripheral blood, with little obvious change in the bone marrow. Nevertheless, it would seem inadvisable to retest her for sensitivity to this drug.

SUMMARY AND CONCLUSIONS

A case of pancytopenia following administration of hydrallazine ("apresoline") for seven months is reported. The patient is making a hematological recovery. Caution should be exercised when administering this drug. Periodic determination of the platelet and white blood cell counts would appear to be desirable.

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MECHANISM OF POISONING FROM WAX CRAYONS

Fredric Rieders, Ph.D.
and
Heinrich Brieger, M.D., Philadelphia

Severe poisoning from ingestion of wax crayons was reported by Brieger and others [1] and discussed in an editorial in THE JOURNAL.[2] Since then, additional cases have come to our attention.[3] Methemoglobinemia was the chief sign of intoxication in all human cases; it was reproduced in one-third of a group of cats fed red and red-orange crayons.[4] The pigment used in certain brands of such crayons was para red, a dye considered insoluble and harmless. Oral or intravenous administration of para red to rats, dogs, and cats has not resulted in methemoglobinemia.[5] However, the intermediate of this dye, *p*-nitroaniline, is a methemoglobin-forming substance.

Since only a small percentage of children who are known to have ingested pieces of crayons have become affected, the following explanations of this type of poisoning were given consideration: (*a*) presence of unreacted amines in some batches of the dye; (*b*) catabolization of the dye to methemoglobin-forming breakdown products due to specific gastrointestinal conditions; and (*c*) individual hypersensitivity. The presence of unreacted *p*-nitroaniline in the finished dye was denied by its chief manufacturer. Since, however, previous investigation had not shown catabolization of the dye in the gastrointestinal tract, samples of para red and of wax crayons containing this dye were examined for *p*-nitroaniline.

Eight different samples of para red toner and 24 different red wax crayons were analyzed. The analysis was performed in the following way: Samples of the wax crayons or the toner weighing 4 gm. each were ground in an agate mortar. The powder was mixed with 50 ml. of 10% sulfuric acid and warmed in a beaker to 50 C for 10 minutes while being stirred. The suspension was filtered through Whatman no. 50 filter paper; the filtrate was made alkaline with sodium hydroxide and extracted with three 50 ml. portions of amyl alcohol. The alcohol was divided into two equal portions. Each portion was evaporated to dryness at room temperature under reduced pressure. The residue from one portion was diazotized with hydrochloric acid and sodium nitrite, coupled with sodium naphthoate, and finally extracted with 20 ml. of benzene; the residue from the second portion was taken up in an equal volume of benzene. The optical densities of the two solutions were measured against a benzene blank in a Beckman D U quartz spectrophotometer at a wavelength of 540 mμ. The benzene was then evaporated, and the residue was tested for para red.[6]

The optical density of the samples that had not been diazotized and coupled with sodium naphthoate did not differ from that of the benzene blank, nor were their dried residues positive for para red. It was thus established that in the extraction procedure outlined above no para red had washed through. A different result was obtained with a number of the samples that were diazotized and coupled with sodium naphthoate. Two of the 8 samples of para red toner and 3 of the 24 wax crayons gave optical densities from 0.180 to 0.310, and tests of their residues, obtained by evaporating the benzene, were positive for para red. Only the presence of unreacted *p*-nitroaniline can explain the synthesis of the pigment. The clinical observations together with the experimental evidence indicate the necessity of careful analysis of pigments prior to their use in the manufacturing of wax crayons.

SUMMARY

Two of 8 samples of para red and 3 of 24 red wax crayons were found to contain unreacted *p*-nitroaniline. Methemoglobinemia following ingestion of red wax crayons is obviously due to the presence of *p*-nitroaniline in certain brands or batches of these crayons.

1025 Walnut St. (Dr. Brieger).

From the Department of Pharmacology and the Division of Industrial Medicine, the Jefferson Medical College of Philadelphia.

1. (*a*) Brieger, H.: Poisoning Due to Ingestion of Wax Crayons, Am. J. Pub. Health **39**: 1023, 1949. (*b*) Jones, J. A., and Brieger, H.: Poisoning Due to Ingestion of Wax Crayons: Report of a Case, J. Pediat. **30**: 422, 1947. (*c*) Murphy, F. J.; Zinzi, F. L., and Murphy, L.: Methemoglobinemia, Clin. Proc. Child. Hosp. **3**: 105, 1947. (*d*) Govan, C. D., quoted by Murphy.[1c] (*e*) Clark, E. B.: Poisoning Due to Ingestion of Wax Crayons: Report of a Case, J. A. M. A. **135**: 917 (Dec. 6) 1947. (*f*) Irwin, H. R., and Barnes, M. W.: Methemoglobinemia Caused by Crayon Ingestion, to be published.

2. Poisoning from Drawing Crayons and Safety Matches, Current Comment, J. A. M. A. **141**: 393 (Oct. 8) 1949.

3. Personal communications to the authors.

4. Spicer, S. S.; Hanna, C. B., and Neal, P. A.: A Note on Methemoglobinemia and Heinz Body Formation in Cats Fed Commercial Crayons, J. Pediat. **33**: 739, 1948.

5. Brieger,[1a] Flinn, F. B.; Axelrod, J., and Brodie, B. B.: The Toxicity of Wax Crayons in Animals, J. Pediat. **33**: 743, 1948.

6. Gardner, H. A.: Physical and Chemical Examination of Paints, Varnishes, Lacquers and Colors, ed. 9, Washington, D. C., Institute of Paint and Varnish Research, 1939, p. 394.

Blindness Due to Glaucoma.—It is estimated by the National Society for the Prevention of Blindness that 12 to 13% of all blindness in the United States is caused by glaucoma. Thirty-four hundred persons become blind because of glaucoma each year while 800,000 persons in the United States are becoming blind from glaucoma and do not know it. Since glaucoma is uncommon before the age of 40 years, and since better medical and public health techniques are continually prolonging the life span, there will be a constantly increasing number of candidates for blindness from glaucoma. Although cataracts are responsible for 20% of all blindness in the United States, surgical treatment can restore useful vision to many patients. A person blinded by glaucoma has an irreversible condition and nothing can be done to restore vision. The blindness is final.—R. E. Prindle, M.D., The Prevention of Blindness Due to Glaucoma, *Postgraduate Medicine*, January, 1953.

COUNCIL ON PHARMACY AND CHEMISTRY

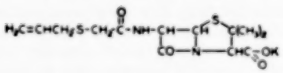
NEW AND NONOFFICIAL REMEDIES

The following additional articles have been accepted as conforming to the rules of the Council on Pharmacy and Chemistry of the American Medical Association for admission to New and Nonofficial Remedies. A copy of the rules on which the Council bases its action will be sent on application.

R. T. STORMONT, M.D., *Secretary.*

Potassium Penicillin O.—Cer-O-Cillin Potassium (Upjohn).—Penicillin O is allylmercaptomethyl penicillin, produced biosynthetically by growing the mold in a medium containing allylmercaptoacetic acid.

Penicillin O is assayed in terms of the International Unit defined as the specific penicillin activity contained in 0.6 γg of penicillin standard. It is stable in dry form at room temperature for a minimum of three years and requires no refrigeration. Solutions may be kept for three days under refrigeration without significant loss of potency. The structural formula of potassium penicillin O may be represented as follows:



Actions and Uses.—Potassium penicillin O has a spectrum of antibacterial activity similar to that of the soluble salts of penicillin G (see New and Nonofficial Remedies under the general statement on penicillin). In experimental animals, penicillin O is found to be less toxic than penicillin G. Absorption and excretion curves of human beings are approximately the same for the two penicillins. Clinically penicillin O has been demonstrated to be as effective as penicillin G and to be less likely to cause sensitivity or allergic reactions. It is particularly useful as a substitute in the treatment of patients sensitive to penicillin G. Physicians should be alert to the development of drug resistant strains. In such instances, therapy should be abandoned in favor of other anti-infective agents.

Allergic reactions to penicillin O have been observed in less than 1% of patients who have no history of previous allergic reactions to penicillin G. Approximately 90% of patients sensitive to penicillin G tolerate therapeutic doses of penicillin O without the development of allergic phenomena. Some patients may lose their sensitivity to penicillin G during a short course of therapy with penicillin O. If reactions occur which cannot be controlled and they are more serious than the condition under treatment, the drug should be discontinued. When administered orally, penicillin O may produce an onion-like odor of the breath which subsides shortly after the drug is discontinued.

Dosage.—Potassium penicillin O is administered orally and by intramuscular injection (intermittent or continuous infusion). In general the dosage is the same as that recommended for penicillin G and therefore varies with the type and severity of the infection. Penicillin O produces blood levels comparable to those obtained with penicillin G administered by similar routes.

By intramuscular injection, a minimum total daily dose of 240,000 units is recommended in most cases. In serious infections, an initial dose of 50,000 to 100,000 units, with a continuing dose of 30,000 to 50,000 units, is injected every three to four hours until the temperature has been normal for forty-eight hours. In traumatic conditions as a prophylactic, doses of 20,000 units every two hours or 30,000 to 50,000 units every three to four hours are administered as an adjunct to local treatment. The total daily dosage may be increased to as high as 2 million units, depending upon the response. For acute gonorrhea, an initial dose of 50,000 units followed by 50,000 units at the end of one hour and 100,000 units at the end of the second hour is recommended.

Oral administration of the drug generally should be reserved for less severe infections. For streptococcal infections without bacteremia, pneumococcal infections, or minor staphylococcal infections without bacteremia, an initial dose of 500,000 units followed by 100,000 units every three hours is recommended. Other penicillin-susceptible infections not involving bacteremia may be treated with similar doses, increased when necessary to 500,000 units every three hours for more severe infections. For acute gonorrhea, the oral dose is 100,000 units every three hours, six times daily for one or two days, or 500,000 units every six hours for three doses. In chronic gonorrhea with complications, a dosage of 500,000 units every four hours should be given. For prophylaxis in tonsillectomy, tooth extraction, and other operative procedures in which secondary infection may occur, the recommended oral dosage is 100,000 units every three hours or 250,000 to 500,000 units every four or five hours given for one day prior to surgery and three to four days postoperatively. The value of oral prophylaxis in rheumatic fever complicated by nasopharyngeal hemolytic streptococcal infections or for exposure to gonorrhea is not yet established. Oral therapy should not be used for meningitis, endocarditis, actinomycosis, and syphilis. Whenever the response to oral medication in other infections is inadequate, the drug should be given parenterally.

The Upjohn Company, Kalamazoo, Mich.

Cer-O-Cillin Potassium: 200,000 unit vials.

Tablets Cer-O-Cillin Potassium (Buffered): 100,000 units. Buffered with 0.25 gm. of calcium carbonate.

Polymyxin B Sulfate (See THE JOURNAL, Nov. 22, 1952, p. 1219).

Chas. Pfizer & Company, Inc., Brooklyn.

Sterile Powder Polymyxin B Sulfate (Topical): 200,000 unit vials. Each vial contains 200,000 units of polymyxin B sulfate equivalent to 20 mg. of polymyxin B standard.

Aminophylline-U.S.P. (See New and Nonofficial Remedies 1952, p. 294).

Taylor Laboratories, Houston, Tex.

Tablets Aminophylline: 0.1 and 0.2 gm., uncoated; 0.2 gm. enteric coated.

Bacitracin (See New and Nonofficial Remedies 1952, p. 41).

Chas. Pfizer & Company, Inc., Brooklyn.

Ointment Bacitracin: 14.2 gm. tubes. An ointment containing 500 units of bacitracin in each gram.

Ophthalmic Ointment Bacitracin: 3.5 gm. tubes. An ointment containing 500 units of bacitracin in each gram.

Cortisone Acetate (See New and Nonofficial Remedies 1952, p. 322).

Merck & Company, Inc., Rahway, N. J.

Suspension Cortone Acetate: 10 cc. vials. A suspension containing 50 mg. of cortisone acetate in each cubic centimeter. Preserved with 0.9% benzyl alcohol.

Dihydrostreptomycin-U.S.P. (See New and Nonofficial Remedies 1952, p. 130).

Chas. Pfizer & Company, Inc., Brooklyn.

Solution Dihydrostreptomycin Sulfate: 2 cc. Steraject cartridges, 2 cc. and 10 cc. vials. A citrate buffered solution containing the equivalent of 0.5 gm. of dihydrostreptomycin base in each cubic centimeter. Stabilized with 1% sodium bisulfite and preserved with 0.25% phenol.

Vitamin K_1 (See New and Nonofficial Remedies 1952, p. 479).

Merck & Company, Inc., Rahway, N. J.

Emulsion Mephyton: 1 cc. ampuls. An emulsion containing 50 mg. of vitamin K_1 in each cubic centimeter.

THE JOURNAL
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SURGICAL ASPECTS OF PEPTIC ULCER

The subcommittee on the "surgical aspects of peptic ulcer" of the Gastroenterological Association presents a critical analysis of the results of the various surgical procedures now used in the treatment of peptic ulcer. In this study the results of vagotomy alone, gastroenterostomy with and without vagotomy, and subtotal gastric resection with and without vagotomy in the treatment of duodenal and gastrojejunal ulcer were compared.[1] This extensive report is based upon 4,076 cases of peptic ulcer. To determine whether the results were influenced by the experience of surgeons in a particular city or by the surgeon's personal experience, the patients were divided into two groups made up of 3,278 patients operated on by surgeons in cities from which more than 50 vagotomies were reported and of 798 patients operated on by surgeons who had performed less than 50 operations of this type. The committee is well aware of the fact that results of any form of ulcer therapy are dependable only when evaluated after at least five years; however, it is pointed out that poor results in a shorter period of time can be interpreted as a definite indication that the particular therapy can be considered a failure.

The larger of the two groups consists of 3,278 cases collected from 110 surgeons, in 72 hospitals, in 12 cities, in the United States and Canada, reporting 50 or more vagotomies in response to a questionnaire sent out by the committee to all general hospitals in these countries in 1948. Fifty-four surgeons in 25 hospitals of this group provided additional follow-up data on their patients one year after the original survey. The data for the large and the small groups were collected between March 1, 1949, and Jan. 1, 1950. The follow-up data on the large group were returned between Jan. 1 and Feb. 12, 1951.

The mortality rate in the patients treated by vagotomy alone varied from 0.4 to 3.2% and did not differ significantly from the mortality following vagotomy plus another surgical procedure (1.0 to 2.2%) with the exception of the small group of gastric ulcers, treated by vagotomy plus gastric resection (7.4%). Persistent diarrhea was reported in 2.7 to 3.8% of cases of vagotomy alone at the time of operation and at least occasional diarrhea in approximately 15 to 30% of patients throughout the period of follow-up. Persistent gastric retention in the postoperative period was reported in 11.7 to 12.6% of the patients who had vagotomy alone for duodenal ulcers, compared to only 3.1 to 4.9% for those having vagotomy plus another surgical procedure. It was necessary to perform other surgical measures after vagotomy alone for duodenal ulcer in 9.7 to 20% of cases, presumably owing to the occurrence of gastric retention. Abdominal distress other than ulcer pain was reported in approximately 35 to 45% of cases after vagotomy alone, compared to approximately 20 to 30% after vagotomy plus another surgical procedure. No other harmful effects in significant numbers were reported, indicating that the only significant harm done by vagotomy is the induction of gastric retention severe enough to be reported as a postoperative complication in a considerable percentage of patients at the time of study.

The data show that subjective and objective improvement may be expected in 81.2% of gastrojejunal ulcer cases subsequent to gastroenterostomy and in 72.1% of gastrojejunal ulcer cases subsequent to gastric resection when these are treated by vagotomy alone. Since the latter type of gastrojejunal ulcer represents a particularly difficult problem in management, vagotomy here appears to be a valuable therapeutic measure. The postoperative occurrence of a significant number (21%) of positive reactions after the insulin test suggests that the gastric vagi were not severed completely in all patients.

The committee concludes that vagotomy alone for duodenal ulcer produces significantly inferior results compared to vagotomy plus gastroenterostomy or vagotomy plus gastric resection. Vagotomy plus pyloroplasty in a relatively small series gave poor results in duodenal ulcer. No significant differences were found between vagotomy plus gastroenterostomy and vagotomy plus gastric resection for duodenal ulcer. Gastric retention was the only significant harmful effect of vagotomy. Good results following vagotomy could not be correlated with physiological evidence of the completeness of the vagotomy.

Vagotomy alone for gastrojejunal ulcer produced definite benefits, and this was particularly significant for those gastrojejunal ulcers following gastric resection. Neither the operative approach (transthoracic or subdiaphragmatic) nor the presence or absence of previous hemorrhage seemed to have a definite effect on the results of vagotomy.

The question of the value of subtotal gastric resection for duodenal and gastrojejunal ulcer was studied in a parallel manner by questionnaires sent to the same surgeons at the same time as the vagotomy follow-up questionnaire. The final results of 140 operating surgeons under 59 surgeons in charge from 12 surgical centers were involved in this study. The validity of the statistical treatment of these data depends on the comparability of the two groups of cases; gastric resection alone and vagotomy with gastroenterostomy or gastric resection. Partial resection alone for duodenal ulcer, when more than 70% of the stomach was removed, has produced results as good as those produced by partial

1. Report of the Committee on Surgical Procedures of the National Committee on Peptic Ulcer of the American Gastroenterological Association on Study of Vagotomy, Study of Gastric Resection, Comparative Study of Vagotomy and Gastric Resection, Gastroenterology **22**: 303-496 (Nov.) 1952.

resection plus vagotomy, with the possible exception of a higher incidence of histamine achlorhydria. It is stressed, however, that further follow-up data will be necessary to confirm this advantage, which may prove to be significant in the prevention of late recurrence of ulcer. It was also shown that partial resection alone is superior in its results to gastroenterostomy plus vagotomy. In all other respects partial gastric resection alone produced significantly better results than vagotomy plus gastroenterostomy. In the treatment of gastrojejunal ulcers following gastroenterostomy the results were better after resection alone than after vagotomy. Although comparable cases were not available, vagotomy alone was superior in the treatment of gastrojejunal ulcer following gastric resection. The mortality after vagotomy alone was significantly lower.

FATAL ACCIDENTS IN THE AMERICAN HOME

When Charles Dickens wrote some decades ago that "accidents will occur in the best regulated families," he presumably was unable to foretell the time when accidents in and about the American home would constitute a major threat to the lives and limbs of its inhabitants. Accidents at home annually kill about 28,000 persons in the United States, with 100,000 persons permanently crippled or disabled, and 4 million more injured sufficiently to remove them from routine activities for one or more days. While many accidents can be attributed to such environmental hazards as inadequate maintenance of household appliances, poor housekeeping, and faulty construction of the house, poor judgment, fatigue, and ignorance of the existence of an unsafe environment contribute greatly to the circumstances surrounding fatal accidents in and about the home.

Data [1] recently obtained from the records of a large group of persons who died as the result of fatal home accidents during 1951 indicate that falls were responsible for nearly one-half of all fatalities. Although falls from chairs, beds, roofs, windows, porches, and ladders contributed substantially to the total, stairs and steps accounted for nearly as many fatalities as all other types of falls combined. A large number of accidents occurred while the victim was walking about a room or walking casually from one room to another. Fire accounted for one-fourth of the fatal injuries among men and for one-third of the total among women, with careless smoking and asphyxiation by smoldering mattresses ignited by cigarettes important contributory factors.

Absorption of poisonous gases, due mainly to the use of gas heaters in poorly ventilated rooms, the seepage of coal gas from stoves and furnaces, leaking gas appliances, inadvertent turning on of stove jets, and the extinction of flames by liquids and foods boiling over also resulted in a substantial number of fatalities in and about the home. Other common causes of death included mishaps with firearms, running an automobile motor in a garage, and acute poisoning by solids or liquids.

One of the outstanding facts to emerge from this study is that a large proportion of the accidental fatalities in the American home are preventable. Since many communities throughout the United States are becoming increasingly aware of the toll exacted by home accidents and are launching campaigns to reduce the loss, physicians everywhere should most heartily encourage their educational efforts.

NUMBER OF PHYSICIANS IN ARMED FORCES REDUCED

At one of President Eisenhower's press conferences, a subject of great importance to the medical profession was mentioned that received only slight recognition in the press. In his brief comments, the President indicated that the armed services had reviewed their requirements for physicians and had reduced the number to be called in the first quarter of 1953 from 1,800 to 1,200. Actually, the reduction in the number of physicians required represents a welcome lowering of the present ratio of physicians from 3.7 to 3.0 per 1,000 troops. The Council on National Emergency Medical Service of the American Medical Association, working with medical advisory committees such as the Health Resources Advisory Committee, has, for many years, persistently striven to effect greater economy and efficiency in the utilization of medical manpower by the armed forces.

The existence of legislation such as the "Doctor-Draft law" as a device for obtaining physicians for military service on an involuntary basis has accentuated the need for more economical utilization of physicians. It is reassuring to note that the all-time high figure of about 6 physicians per 1,000 troops, which was reached during World War II, has now been cut in half. This means that fewer physicians will be disrupted in their civilian practice and, as a result, the continuation of the present high standard of civilian medical care will be insured. Inasmuch as the medical accomplishments of the military services since the beginning of the Korean war are the best in the history of the United States, it is apparent that the reduction in the ratio of physicians in service has in no way impaired the quality of medical care in the armed forces.

POSTGRADUATE MEDICAL EXPENSE AND INCOME TAXES

For some time the House of Delegates, the Board of Trustees, and physicians generally have been concerned over the disallowance for federal income tax purposes of postgraduate medical expenses. Recently, similar expenses incurred by a lawyer, Coughlin, were disallowed. Because of the similarity of the issues involved, the American Medical Association entered this case as amicus curiae. On April 14, the U. S. Court of Appeals, Second Circuit, held that the expenses incurred by Coughlin were deductible because, in part, a lawyer has a moral and professional duty to keep sharp the tools he uses in his practice. Further comment on the implications of the Coughlin case will appear in a subsequent issue of THE JOURNAL after the court opinion has been studied.

1. How Fatal Home Accidents Occur, Statist. Bull. Metrop. Life Insur. Co. 34:6 (Feb.) 1953.

THE PRESIDENT'S PAGE

A MONTHLY MESSAGE

The day after the meeting of the House of Delegates in Washington, I left for Europe. I visited Holland, England, Portugal, Italy, and France. Most of my time was spent in Portugal at the meeting of the Council of the World Medical Association. I should like to feel that all the readers of this page know what the World Medical Association is and stands for, but unfortunately I know that many do not. Even many of those who do know of my interests in it will nine times out of ten ask me how the World Health Organization is doing.

So let me start off by distinguishing between the two organizations. The World Health Organization is a branch of the United Nations and represents the governments of the world in the field of health. It is supported entirely by government funds. On the other hand, the World Medical Association is an organization of the national medical associations and represents the practicing profession of the world. It is supported entirely by dues and voluntary contributions. There is only one member per country, and that is the national medical association of the country that is most representative of the medical profession in that country. The American Medical Association is the American member. Forty-three nations are now members of the association. The membership does not include any country behind the iron curtain.

The aims of the World Medical Association are to effect a better liaison among the doctors of the world; to serve as a forum for discussion of mutual problems; to disseminate information; to raise the standards of health, medical education, and medical care throughout the world; to represent the practicing profession before other international bodies, when matters of health and medicine are discussed, and to give the average doctor a voice in international affairs; and to improve international relations.

Why should the average doctor have any interest in the organization? I believe there is more than one reason why he should. First of all, an increasing number of problems relating to health and medicine are being discussed and decided on an international level. Most of the bodies discussing these problems have one viewpoint only, and that is the viewpoint of government. Without the World Medical Association, there is no one to present the opinions of practicing physicians. Their views should be heard and considered, but they cannot give them as individuals. Their voice can be heard only through an organization. The World Medical Association, through its 43 member associations, can speak for 700,000 physicians. I believe all will agree that this is a voice that should not be ignored. Furthermore, what happens in other countries is sooner or later likely to affect us here. Therefore, we should work with our colleagues in other countries and attempt to solve the problems facing the profession.

Six years of experience with doctors of other nations has shown me conclusively that the same problems face the profession in all countries. They differ only in degree. That, perhaps, is not surprising. What is surprising is that doctors all over the world think alike about these problems, differing only as to details.

We are indeed fortunate in the United States that government has not made the inroads on the practice of medicine that it has in many other countries. Canada and the United States are the freest countries in the world as far as medical practice is concerned, and both countries intend to keep it that way. One way we can help to accomplish that objective is by cooperating with other countries and fighting the inroads of socialism via the international route. The World Medical Association made a great contribution to this fight by its publicity on the machinations of the International Labor Organization. That one action alone warrants the support of the doctors of America. (See THE JOURNAL, May 31, 1952.) The association also protested some of the socialistic propositions in the Document on Human Rights.

Another great contribution to the welfare of medicine by the World Medical Association is the sponsorship of the First World Conference on Medical Education, to be held in London, April 22-29, 1953. Medical Education is long overdue for a reassessment. Medicine has changed greatly, owing to new methods of diagnosis and treatment. The visual aids in medical education are comparatively new, yet bid fair to revolutionize teaching. The didactic lecture seems to be on the way out. The textbook has to a certain extent outlived its usefulness. Bedside teaching has come more and more to the fore. Television permits closer observation of the operating field than the old amphitheater ever could. Yet, medical education is still largely in the hands of those brought up under the old system.

This conference will give the practicing physician an opportunity to state how medical education has met or failed his needs. No previous conference, even on a local basis, has given consideration to the ideas of the doctor who is not a medical educator, yet is the consumer of medical education. I outlined the main theme and the subtopics to be discussed in my President's Page for November, 1952. They are also given in THE JOURNAL in an editorial, March 7, 1953, page 822.

No one, of course, expects that any uniform pattern of medical education will develop from the conference. Medical education must conform to the local needs, the facilities, and the economics of the various countries; however, an exchange of ideas will be beneficial, and a general goal at which to aim may well result. The proceedings will be published and will form the most valuable contribution to a survey of medical education ever presented.

The World Health Organization, the Council of International Organization of Medical Sciences, and the International Association of Universities will participate in the conference. Invitations have been sent to all medical schools, national medical associations, and other organizations interested in medical education. Professor Sir Lionel Whitby, Vice Chancellor of the University of Cambridge, will preside over the conference.

This one event will further have justified the existence of the World Medical Association. In addition to this, its studies in the field of the medical aspects of social security, standards of hospitals, the development of an international code of medical ethics, its stand with reference to an international pharmacopeia, and its continuing fight to protect the rights and privileges of doctors in both peace and war are further examples of its importance. Most important of all, however, is the ability of the World Medical Association to speak for the practicing profession. Too often the profession is ignored in deciding matters of moment to both the profession and the public.

While no individual can belong to the World Medical Association, yet in the United States, and more recently in some other countries, supporting committees have been established in which individual membership is possible. In the United States we have established the World Medical Association, United States Committee, Inc., a nonprofit organization. Medical societies, business organizations, and individuals may become members. All members receive the bulletin of the association, a quarterly publication giving news of the association and medical news of the world; all other publications of the association; a membership card; and letters of introduction facilitating visits to medical centers in other countries. Committee members may attend the general assemblies of the association as official observers.

I should like to see every member of the American Medical Association a member of the United States committee. In that way we can have an effective voice in international medical affairs, a voice that is sadly needed, and we can make the World Medical Association a powerful influence in molding world opinion about medicine.

LOUIS H. BAUER, M.D., Hempstead, N. Y.

ORGANIZATION SECTION

REPORT OF BOARD OF TRUSTEES

The following report of the Board of Trustees will be presented to the House of Delegates of the American Medical Association at its session to be held in New York in June:

FINANCIAL STATEMENT

The official reports of the Treasurer and of the Association's auditors for 1952 are appended as a part of this report.

Collections of 1952 American Medical Association membership dues, through the constituent state and territorial medical associations, amounted to $2,980,000. This is well in excess of last year's collections and proves beyond question that the Association's membership dues program has been gaining wide support. A comparative study of the figures indicates, too, that secretaries of state medical societies were most diligent in their efforts to collect delinquent dues for 1950 and 1951. They succeeded in bringing $58,874 in delinquent dues into the membership dues fund in 1952.

Subscription receipts from the Special Journals amounted to $508,269, approximately $41,000 more than in 1951.

Advertising income showed a substantial gain over 1951. THE JOURNAL increased its advertising income $579,701 to $3,008,911 in 1952, and the combined increase in advertising revenue from the Special Journals and *Today's Health* amounted to $16,010.

Fellowship dues were discontinued at the end of 1951, thus eliminating this source of income, which last year approximated $229,000.

Income from Association investments amounted to $197,254, about $30,000 more than in 1951. The portfolio at the year's end, evaluated at cost, amounted to $7,081,035 compared to $6,542,862 in 1951. Income was augmented by the higher return on equities, which composed a larger share of the portfolio in 1952. The over-all return on investments was about 2.8% in 1952 and 2.5% in 1951. A substantial portion of Association funds continues to be invested in government securities that carry low yields.

Miscellaneous receipts approximated $68,000. Gross income totaled $8,213,341.

Printing and publication costs of periodicals, books, and pamphlets totaled $4,101,541. Comparable costs in 1951 were $3,734,184. The approximate 10% increase in costs over 1951 is ascribed to larger production, higher prices for paper and materials, and continuation of a trend toward higher wage rates that has been evident for several years in the production and office divisions. Total wages paid to an average number of 850 employees in 1951 in both divisions amounted to $3,329,109 compared with wages of $3,706,507 paid to an average number of 875 employees in 1952. Approximately 5,500 tons of paper were used in all printing operations of the Association in 1952. This was valued at $975,000, compared with 5,250 tons in 1951 and valued at $875,000.

Expenditures of Councils, Bureaus, and related activities aggregated $2,769,405, an increase of $525,525 over 1951. The increase is due in part to added projects, to expanded activities of the Council on Medical Education and Hospitals, the Department of Public Relations, Membership Records, the Bureau of Exhibits, and others, and again to high operational costs. Expenditures of each Council, Bureau, and related activity for the year 1952, together with a consolidated classification of the expenses, appear in the auditor's report.

Employees' past service annuities and retirement pay amounted to $59,271 and $135,126, respectively, in 1952 and 1951. Purchase of past service annuities, amortized over the last 9 years, was completed in 1952. Current annuities are being purchased for employees on a contributory basis.

The annual and interim meetings of the Association were conducted at a net expense of $85,376, compared to $79,739 in 1951. Cash discounts of $49,522 were allowed to advertisers over the amount received from vendors, compared with $37,701 in 1951. Depreciation and taxes on headquarters office equipment amounted to $24,763, compared with $28,329 in 1951.

Legal and sundry expense amounted to $67,790. After three years of litigation, the Hoxsey suit was brought to a close at an expense to the Association of $44,816, which is included in the total.

Expenditures of the National Education Campaign amounted to $255,192 compared to $529,514 in 1951. Campaign activities terminated on Sept. 18, 1952.

For the third consecutive year $500,000 was appropriated out of income to support the American Medical Education Foundation.

Capital expenditures during 1952 for equipment and building improvements approximated $65,000. At the end of 1952, Association properties and equipment had a net book value of $1,788,549, equal to 54% of cost.

Marketable securities on Dec. 31, 1952, valued at cost, amounted to $5,587,507 exclusive of $1,493,529 in securities held in the American Medical Association Research Fund. This total figure, $7,081,036, compares with securities totaling $6,542,863 at the end of 1951.

The following changes have been made in Reserve Funds: the Retirement Reserve Fund decreased to $150,000 from $675,000; the Building Reserve Fund increased from $450,000 to $1,000,000, and the Depreciation Reserve Fund increased from $1,455,000 to $1,540,000.

No changes have been made in the Association Reserve Fund, $350,000; the Equipment Modernization Reserve Fund, $700,000; the American Medical Education Foundation Reserve Fund, $500,000; or the American Medical Association Research Fund, $1,494,075.

TREASURER'S REPORT

REPORT OF THE TREASURER OF THE AMERICAN MEDICAL ASSOCIATION FOR THE YEAR ENDED DEC. 31, 1952

Investments (at cost as of Jan. 1, 1952)		$5,049,333.83	
Bonds purchased (at cost)	$1,700,507.09		
Common stocks purchased (at cost)	295,028.31	1,995,535.40	
		7,045,369.23	
Less:			
Bonds called, matured, or sold	$1,399,371.40		
Common stocks sold	58,190.94	1,457,562.34	
Investments as of Dec. 31, 1952			$5,587,506.89
Balance held for investment:			
Jan. 1, 1952	$ 35,635.51		
Interest and dividends received in 1952	197,167.29		
	$ 232,802.80		
Transferred to general fund	157,028.75		
Uninvested funds as of Dec. 31, 1952			75,774.05
Invested and uninvested funds as of Dec. 31, 1952			$5,663,280.94

AMERICAN MEDICAL ASSOCIATION RESEARCH FUND

Investments (at cost) as of Jan. 1, 1952		$1,490,528.76
Uninvested funds as of Jan. 1, 1952		546.07
Invested and uninvested funds as of Dec. 31, 1952		$1,494,074.83
Interest received on investments and transferred to general fund to apply on research expenditures	$ 37,028.75	

DAVIS MEMORIAL FUND

Funds on deposit at Jan. 1, 1952	$ 8,528.14	
Interest earned on bank balance in 1952	128.40	
Funds on deposit at Dec. 31, 1952		$ 8,656.54

JOSIAH J. MOORE, Treasurer.

AUDITOR'S REPORT

March 27, 1953

The Board of Trustees
American Medical Association:

We have examined the balance sheet of American Medical Association as of Dec. 31, 1952, and the related statement of income for the year then ended. Our examination was made in accordance with generally accepted auditing standards, and accordingly included such tests of the accounting records and such other auditing procedures as we considered necessary in the circumstances.

In our opinion, the accompanying balance sheet and statement of income present fairly the financial position of the American Medical Association at Dec. 31, 1952, and the results of its operations for the year then ended, in conformity with generally accepted accounting principles applied on a basis consistent with that of the preceding year.

PEAT, MARWICK, MITCHELL & Co.

STATEMENT OF INCOME

FOR THE YEAR ENDED DEC. 31, 1952

Income:		
Membership dues, of which $58,874 applies to prior years, and subscriptions to periodical publications		$4,695,819
Advertising		3,282,429
Income from investments		197,254
Miscellaneous receipts and other income		67,839
		8,243,341
Expenses:		
Printing and publication costs of periodicals, books and pamphlets	$4,101,541	
Councils, bureaus, and related activities (schedule 1)	2,769,465	
Employees' past service annuities and retirements	59,272	
Annual and interim sessions—net	85,376	
Cash discounts—net	49,522	
Depreciation and taxes—unallocated	24,763	
Legal and sundry	67,700	
	7,157,609	
Expenditures for National Education Program	255,192	
Appropriated for The American Medical Education Foundation	500,000	7,912,801
Income in excess of expenses		$ 330,480

Balance Sheet—Dec. 31, 1952

Assets

Cash			$ 104,582
Accounts receivable:			
Advertising	$ 290,804		
Directory report service—19th edition	26,057		
Other	93,891		388,752
Interest accrued on investments			34,275
Inventories of materials, supplies, work in progress, and publications, at cost			466,595
Expenditures on publication in progress			103,059
Prepaid expenses, deposits and advances:			
Deposits and advances		67,768	
Insurance and other prepaid expenses		39,554	107,322
Marketable securities at cost (value based on market quotations, $5,525,933):			
United States government securities		3,119,657	
Railroad, municipal, public utility, and industrial bonds		1,802,809	
Common stocks		665,041	5,587,507
Representing investments of:			
The American Medical Education Foundation Fund	$ 500,000		
General fund	1,347,507		
Depreciation reserve fund	1,540,000		
Association reserve fund	350,000		
Retirement reserve fund	150,000		
Building reserve fund	1,000,000		
Equipment modernization reserve fund	700,000		
American Medical Association Research Fund:			
United States government securities at cost (value based on market quotations, $1,482,607)		1,493,529	
Cash		546	1,494,075
Property, plant and equipment, at cost:			
Land		328,774	
Building	2,144,719		
Machinery and printing equipment	706,392		
Office and laboratory equipment	474,810		
	3,325,921		
Less allowance for depreciation	1,537,372	1,788,549	2,117,323
			$10,403,490

Liabilities

Accounts payable and accrued expenses:			
State Journal Advertising Bureau			$ 54,321
Other accounts payable			172,119
Accrued payroll			53,315
Accrued taxes			69,811
Employees' withheld taxes			49,367
			398,943
Provision for payment to The American Medical Education Foundation			500,000
Provision for completion costs of publications to be issued			218,149
Deferred credits:			
Unexpired subscriptions to publications		$ 448,184	
Income from 19th edition directory report service less accumulated costs applicable to directory		121,343	
Other, including 1953 dues of $25,700 received in advance		35,489	605,016
American Medical Association Research Fund reserve			1,494,100
Other reserves:			
Association		350,000	
Retirement		150,000	
Building		1,000,000	
Equipment modernization		700,000	2,200,000
Capital account:			
Balance at Dec. 31, 1951	$4,711,892		
Less net amount transferred to other reserves	25,000	4,686,892	
Excess of income over expenses for year ended Dec. 31, 1952		300,480	4,987,382
			$10,403,490

EXPENSES OF COUNCILS, BUREAUS, AND RELATED ACTIVITIES

FOR THE YEAR ENDED DEC. 31, 1952

Salaries and wages	$1,310,304
Office printing and supplies	149,474
Postage, express, and freight	70,728
Telephone and telegraph	16,713
Memberships, books, and periodicals	33,922
Legislative services	5,963
Statistical equipment rental	28,352
Membership records and reimbursement to state societies for dues collections	141,054
Membership conversion	86,243
Biographical records	105,881
Staff and officers' travel	128,487
Inspection of hospitals and medical schools	28,842
Joint commission on hospital accreditation	28,500
General administrative expenses including allocation of headquarters maintenance, employee insurance, and taxes	227,528
Educational exhibits, motion pictures, and literature	133,567
Radio and television	94,754
Trustee meetings	13,000
Committee conferences	214,905
Grants, tests, and surveys	22,478
Miscellaneous	42,439
	$2,769,405

Note:—The above expenses are allocated to the following Councils, Bureaus, and related activities:

Association activities	$ 154,809
Secretary's office	81,336
Association trustees	9,786
Membership records	141,054
Membership conversion	86,243
Biographical records	105,881
Survey of Medical Education	51,632
Commission on Chronic Illness	20,000
Student American Medical Association	23,789
American Medical Education Foundation	44,100
Department of Public Relations	285,048
Washington Office of A. M. A.	175,643
Chemical laboratory	70,461
Microbiologic laboratory	18,587
Council on Medical Education and Hospitals	297,489
Council on Medical Service	111,239
Council on Pharmacy and Chemistry	127,775
Council on Physical Medicine and Rehabilitation	74,791
Council on Foods and Nutrition	48,154
Council on Industrial Health	72,010
Council on National Emergency Medical Service	36,493
Council on Rural Health	43,288
A. M. A. Library	61,734
Bureau of Exhibits	81,503
Bureau of Health Education	221,857
Bureau of Investigation	20,087
Bureau of Legal Medicine and Legislation	57,756
Bureau of Medical Economic Research	169,182
Committee on Cosmetics	16,316
Committee on Mental Health	9,321
Committee on Research	48,240
	$2,769,405

STUDENT CONVENTION IN CHICAGO

The third annual convention of the Student American Medical Association will be held June 15-17 at the Edgewater Beach Hotel, Chicago. More than 1,000 registrants are expected to attend. The tentative schedule announced by President David Buchanan, University of Illinois senior, calls for a business session of the House of Delegates on Monday. Abbott Laboratories will be hosts at an informal get-together that night.

Tuesday morning will feature three panels devoted to the "doctor draft," general practice, and S. A. M. A. chapter activities. In the afternoon tours will be conducted through A. M. A. Headquarters. On the final day will be the election of officers and a première of an outstanding medical film. This year a technical exhibit will be on display for the first time at an S. A. M. A. convention. All A. M. A. members are invited to attend.

STATEMENT BY ELMER L. HENDERSON, M.D., BEFORE SENATE AND HOUSE COMMITTEE ON REORGANIZATION PLAN NO. 1, 1953

Gentlemen: My name is Elmer L. Henderson. I am chairman of a special liaison committee of the Board of Trustees of the American Medical Association and also am a former president of the Association. Accompanying me today are Doctors Edward J. McCormick, President-Elect of the Association; Dwight H. Murray, Chairman of the Board of Trustees; Leonard W. Larson, a trustee, and John W. Cline, immediate past president of the Association.

I am appearing here today at your invitation to present the position of the Association relative to Reorganization Plan No. 1 of 1953. With your kind indulgence, I should like to trace very briefly the history of the position of the American Medical Association concerning the establishment of a separate governmental department in charge of health affairs.

As early as 1884, at the annual meeting of the House of Delegates, it was urged that a separate Department of Health be established under a cabinet officer. In 1891 a committee of twenty-three members was appointed to petition the Congress on this matter. Further pertinent references appear in the Proceedings of the House of Delegates for each year from 1891 up to and including 1902.

The subject was discussed annually from 1906 to 1913 and from 1917 to 1930. Periodically during the succeeding twenty-two years the House of Delegates has reaffirmed its position in this regard. This position was most recently expressed in detail in a resolution adopted in December 1950 which provided:

"*Resolved*, That the House of Delegates of the American Medical Association urges all officers and members of the American Medical Association and all constituent state associations to take immediate steps to implement the passage into law of a bill providing for the coordination and integration of all federal health activities under an independent agency with executive status except the medical services of the Armed Forces and the Veterans Administration."

On last Saturday, March 14, the House of Delegates of the American Medical Association met in special session to consider Reorganization Plan No. 1. We were singularly honored by the appearance of the President of the United States, who outlined briefly the position of the Administration with respect to governmental medical affairs. In addition, Senator Robert A. Taft and Congressman Walter H. Judd explained and discussed the details of Reorganization Plan No. 1 and the purposes for which it had been submitted.

In summing up the final action of the Association in this matter, I should like to read a report of the Board of Trustees which was adopted unanimously by the House of Delegates.

Report of Board of Trustees on Reorganization Plan No. 1 of 1953, Adopted Unanimously by the House of Delegates on March 14

The House of Delegates of the American Medical Association has for nearly 80 years been on record as favoring an independent Department of Health in the federal government. The reason for this stand has been that the House has felt that health and medicine should be given a status commensurate with their dignity and importance in the lives of the American people, and that they should be completely divorced from any political considerations.

The Board of Trustees, after a careful study of the policy of the American Medical Association with respect to the administration of health activities in the Executive Branch of the government and after studying the Reorganization Plan for elevation of the Federal Security Agency to cabinet status submitted by President Eisenhower to the Congress, finds that Reorganization Plan No. 1 of 1953 provides for a special assistant to the Secretary for Health and Medical Affairs. This provision is a step in the right direction which should result in centralized coordination under a leader in the medical field of the health activities of the proposed department. Health, therefore, is given a special position. The proposed plan, properly administered, will permit more effective coordination and administration of the health activities of the new Department without interference or control by other branches.

Previous attempts to raise the Federal Security Agency from an independent agency to the level of an Executive Department have been opposed by the Association because the plan did not meet these aims.

Inasmuch as federal health benefits and programs are established by the Congress, an administration bent on achieving the nationalization of medicine cannot reach that goal except with the support of Congress. Therefore, an organizational plan through which federal health activities are administered, although important, is not nearly so vital an issue as the policies adopted by the Congress of the United States.

The Board of Trustees recommends that the House of Delegates reaffirm its stand in favor of an independent Department of Health but that it support the Reorganization Plan No. 1 of 1953 as being a step in the right direction; that the American Medical Association cooperate in making the plan successful and that it watch its development with great care and interest.

It should be understood, however, that the Association reserves the right to make recommendations for amendment of the then existing law or to press for the establishment of an independent Department of Health, if the present plan does not, after a sufficient length of time for development, result in proper advancement in and protection of health and medical science and in their freedom from political control.

EIGHTH NATIONAL CONFERENCE ON RURAL HEALTH

Successful methods of bringing improved medical care and better health to rural areas were discussed by more than 600 physicians and representatives of agricultural, university extension, public health, and community groups attending the Eighth National Conference on Rural Health at Roanoke, Va., Feb. 27 and 28. The two-day meeting, sponsored by the American Medical Association Council on Rural Health, featured speakers from all sections of the country. Their audience was comprised of rural leaders from 42 states and the District of Columbia.

Dr. Louis H. Bauer, of Hempstead, N. Y., President of the American Medical Association, assured the conference that the A. M. A. will "do everything within its power to help you in your campaign to improve rural health conditions." Dr. Bauer said that no one organization can do the job by itself, because the task requires community effort.

According to Dr. Bauer, "One of the difficulties in the past has been the development of some means to attract physicians to those areas lacking them. A physician who has spent 8 to 13 years of his life in being trained to practice modern medicine is reluctant to settle in an area where facilities are limited. Increasing the number of physicians is not the answer. They would still tend to congregate in cities. In some states, the problem has been solved by the community providing medical facilities and then permitting the physician to rent those facilities or buy them through gradual amortization. Where this has been done, physicians have been obtained." Dr. Bauer added that, because of good roads and automobiles, every community does not need a hospital so long as one is within reasonable distance. Also, today's good transportation facilities permit a physician to cover a much wider territory and to care for more persons.

Stressing the theme of the conference, "Widening the Highway to Health," Dr. F. S. Crockett, of Lafayette, Ind., Chairman of the Council on Rural Health, declared that rural health promotion has become truly a citizenship responsibility. He stated: "No community problem can be solved by a minor segment of the population. We believe we should take advantage of the strength accruing from group action. Citizen organizations lend themselves well to such things as health promotion." He reported that group strength has been obtained through the establishment of rural health councils, made up of all facets of community life. These councils, he pointed out, in many instances have brought about the elimination of health hazards and have taught simple health rules to improve conditions.

How civic enterprise and cooperation induced a young physician to practice in the small community of Huddleston, Va., was reported by Earl J. Shiflet of Richmond, state deputy of the Virginia State Grange. With the help of the state grange, the Bedford County Medical Society, and the Virginia Council on Health and Medical Care, the people of Huddleston prepared for his reception by providing him with a clinic and residence at a reasonable rental. In less than six months, the doctor was treating 250 to 300 patients each week.

At the second session of the opening day, Dr. Carll S. Mundy of Toledo, Ohio, vice-chairman of the Council on Rural Health, spoke on financing rural medical care. One of the great problems of group health and accident insurance in rural areas is enrollment of participants. It is necessary for each community to determine its own health care plans, and Dr. Mundy suggested that farm organizations and church groups might provide the basis for group insurance coverage. "The increasing cost of illness over the past two or three decades has made budgeting against the cost of major illness a necessity for most of our population. As a result, many plans and methods have been devised to meet this need. Those

Dr. F. S. Crockett of West Lafayette, Ind., checks the registration from his home state at the Rural Health Conference with Mrs. Charles W. Sewell of Otterbein, Ind., advisory member of the Council on Rural Health. Dr. Crockett is chairman of the Council.

that have embraced the principles of group coverage have met with the greatest success and have been able to furnish the best coverage at the lowest premium rates. To get the most economical coverage, it is mandatory that all such schemes adhere to certain basic and fundamental insurance principles. None should reward for illness," he said.

Dr. Harold J. Noyes, dean of the University Dental School, Portland, Ore., advised the conference that good dental health service in rural areas requires the combined planning, cooperation, and support of the community. "If the rural areas are to compete with those of the larger cities in their bid for dentists," he said, "it will be necessary to provide these men with the opportunity to administer dental treatment as a true health service and not on a basis of selling their wares over the counter upon the prescription of the patient, who is in no way qualified to make his own diagnosis or prescribe the expedient treatment." For better rural dental care, Dr. Noyes recommended getting farm youth interested in the study of dentistry so that they will return to their home areas to practice, support state department of public health activities and the state dental school, and support the local dentist.

Reiterating the need for financing rural health care outlined by Dr. Mundy, Frank W. Peck of Chicago, managing director

of the Farm Foundation, warned that welfare economics takes over when families do not learn to provide for their own necessities. According to Mr. Peck, the "missing item in the family budget" is medical care, and he recommended education to correct this omission. He added: "I think it is a cooperative job of education. This includes the appeal to logic and reason. It represents the teaching of values and benefits. It represents facing the economic facts of life."

Dr. Paul D. Sanders, of Richmond, Va., editor of the *Southern Planter*, said that many of the health problems of the rural South are due to the tendency of doctors, dentists, and nurses to settle in the cities. He appealed for sympathetic support of the farm family while it develops an economy sufficient to attract physicians to rural areas.

Featured speaker on the Friday evening program was Mr. R. E. Short, of Brinkley, Ark., recently appointed director of the foreign agricultural service, U. S. Department of Agriculture, and former vice-president of the American Farm Bureau Federation. "An expansion of agricultural research is needed to assure adequate dietary and health standards for the people of the United States," he said. "By producing efficiently and abundantly at fair prices, the farmers will have more economic opportunity to take advantage of medical and health facilities."

How medical scholarships have been effective in bringing physicians to rural areas was reviewed by Dr. Felix J. Underwood, of Jackson, Miss., executive officer of the Mississippi State Board of Health. In six years the state of Mississippi awarded 406 medical school scholarships with the provision that the recipient practice medicine after graduation in an approved rural community for a minimum of two years.

Dr. Benjamin N. Saltzman, of Mountain Home, Ark., related his experiences in practicing medicine in a rural community. He recommended that communities fulfill their promises to general practitioners invited to reside in the area. He urged also that either the doctor or the community provide a hospital for maximum medical care.

Community initiative brought better health conditions to Clinton County, reported Dr. Edmond K. Yantes, of Wilmington, Ohio. Two years ago a survey revealed that the area lacked basic standards necessary to safeguard health. "The results of the survey astounded us and awoke in many the desire to improve the situations found. We realized that this was just the beginning of a long-term job, but we found that we had made a great many people health conscious and more susceptible to suggestions for improving them."

Dr. Yantes said the survey brought the desired action and today new doctors have been attracted by the building of a general hospital; infant mortality has been decreased; unsanitary conditions have been reduced; immunization of children against smallpox and diphtheria has reached approximately 90%; milk pasteurization has been instituted; and children entering school are being given check-ups.

Before the two-day conference, on Feb. 26, a "For Doctors Only" session was held. This meeting was devoted to all phases of physician participation in activities at the community level and was followed by an informal discussion of rural health programs and problems.

PROMOTION OF "YOUR DOCTOR" FILM

Most valuable in public relations work of state and county medical societies is the "Your Doctor" film. Many local societies encourage commercial theaters to book the film through RKO-Radio Pictures and by arranging showings of the 16 mm. version throughout the community. Brief announcements of the availability of the film will stimulate community interest. There are two ways in which the 16 mm. version may be booked. Prints are available on loan from Modern Talking Picture Service, Inc., 45 Rockefeller Plaza, New York 20, N. Y. The only charge is for postage and insurance. Individual prints may be purchased at $70 per copy from Arthur M. Good, RKO-Radio Pictures, 1270 Avenue of the Americas, New York, N. Y., and requests will be filled by society offices.

FEDERAL MEDICAL LEGISLATION

Commission on Governmental Functions and Fiscal Resources

Senator Taft (R., Ohio) in S. 1514 and Congressman Halleck (R., Ind.) in H. R. 4406 propose to establish a commission on governmental functions and fiscal resources in line with the recommendation made by President Eisenhower in a special message to Congress. The commission would be composed of 25 members, 15 of whom would be appointed by the President, 5 by the President of the Senate, and 5 by the Speaker of the House. Three members would come from the majority party and two from the minority party in the Senate and House appointees. The commission would study and investigate all the activities in which federal aid is extended to state and local governments to determine whether there is justification for such federal aid, whether there are other fields in which the federal government should participate, whether federal control should be limited, and whether the federal aid should be limited to cases of need. The ability of the federal government and the states to finance such activities would be studied. The commission would submit a final report including legislative recommendations not later than March 1, 1954. Interim reports could be made. This measure was referred to the Committees on Government Operations.

Western Interstate Commission for Higher Education

Senators Hunt (D., Wyo.), Anderson (D., N. Mex.), Barrett (R., Wyo.), Bennett (R., Utah), Chavez (D., N. Mex.), Cordon (R., Ore.), Jackson (D., Wash.), Johnson (D., Colo.), Magnuson (D., Wash.), Mansfield (D., Mont.), Morse (R., Ore.), and Murray (D., Mont.) in S. 1515 would give congressional consent to the states of Arizona, California, Colorado, Idaho, Montana, Nevada, New Mexico, Oregon, Utah, Washington, Wyoming, and the territories of Alaska and Hawaii to enter into a compact relating to higher education. Under the Constitution, interstate compacts are forbidden without the express permission of Congress. Any five or more of the above-mentioned states or territories would be authorized through a commission to cooperatively finance and arrange for students to obtain college courses of technical professional and graduate training, particularly in medicine, dentistry, public health, and veterinary medicine. The commission would consist "of three resident members of each compacting State or Territory." It would make agreements with educational institutions to provide graduate and professional education for the citizens of the compacting states and territories. Reports would be made to the Western Governor's Conference and to the legislatures of the states and territories involved. Recommendations would be made to the governors on uniform legislation dealing with higher education in the region. This bill was referred to the Labor and Public Welfare Committee.

Shorter Periods of Service Under Doctor Draft

Representative Rees (R., Kan.) in H. R. 4416 would reduce the obligatory service period under the doctor draft for physicians in priority 2 who have had less than 21 months' service to "(A) fifteen months if he has had at least twelve but less than fifteen months of such active duty, (B) twelve months if he has had at least fifteen but less than eighteen months of such active duty, or (C) nine months if he has had at least eighteen months of such active duty." Identical with H. R. 3706 (Patterson) previously reported. This measure was referred to the Armed Services Committee.

Professional Education Expense Deductions from Taxable Income

Congressman Davis (D., Ga.) in H. R. 4393 proposes to allow deduction of reasonable costs of professional educational expenses for additional education, if for the purpose of carrying on a profession, improving professional qualifications, or increasing professional remuneration. The present tax laws permit taxable income deductions for ordinary expenses in trade or business, but there is no provision to permit professional persons to deduct the cost of additional education. This bill was referred to the Ways and Means Committee.

The summary of federal legislation was prepared by the Washington Office of the American Medical Association.

MEDICAL NEWS

ARIZONA

State Medical Meeting in Tucson.—The Arizona Medical Association, Inc., will hold its annual meeting April 26-29 in the Pioneer Hotel, Tucson, at the call of its president, Dr. Thomas H. Bate, Phoenix. Dr. Edward M. Hayden, Tucson, will assume the presidency during the general session Monday morning. Guest orators and their first presentations include:

Paul C. Aebersold, Ph.D., Oak Ridge, Tenn., Atomic Energy—National Defense Plus Peace Time Benefits.
Mr. Mac F. Cahal, Kansas City, Mo., The Public Is Your Patient.
Herbert H. Davis, Omaha, Breast Lesions as Seen in the Physician's Office.
Ross Golden, New York, Some Studies of Gas Distention of the Small Intestine.
Samuel M. Feinberg, Chicago, Role of the Nonspecialist in the Care of Allergic Diseases.
John V. Goode, Dallas, Texas, Surgery of the Aged.
John B. Grow, Denver, Exploratory Thoracotomy in the Management of Obscure Lesions of the Chest.
Edmund W. Overstreet, San Francisco, Some Modern Aspects of Estrogen Therapy in Women.
H. Relton McCarroll, St. Louis, Vitamin Resistant Rickets.
Travis Winsor, Los Angeles, Management of Peripheral Arterial Occlusive Disease.
Peter F. Salisbury, Los Angeles, Clinical Experiments with the Artificial Kidney and Related Procedures.
Charles H. Slocumb, Rochester, Minn., Rheumatoid Arthritis, Present Concepts and Treatment.

The Woman's Auxiliary to the association will meet simultaneously at the Pioneer Hotel. The president's dinner dance ($7 per person) Wednesday, 7:45 p. m., will be preceded by a social hour. The golf tournament will be held at the Tucson Country Club Sunday afternoon. Among the organizations that will hold special society luncheon meetings at the hotel during the sessions are the Arizona Rheumatism Association, Sunday, 12:30 p. m.; Arizona Chapter, American College of Surgeons, Monday, 12:30 p. m.; and Arizona County Medical Society Secretaries, Monday, 12:30 p. m. On Tuesday the Arizona Society of Allergy, meeting at 12 noon, will have as guest Dr. Samuel M. Feinberg. The Arizona Chapter, American Academy of General Practice (12 noon) will hear Mr. Mac F. Cahal on "Academy Program in the Elevation of Standards in General Practice and the Expansion of Facilities for Postgraduate Training." The Arizona Radiological Society (12:30 p. m.) will have as guests Paul C. Aebersold, Ph.D., and Dr. Ross Golden; and at 12:30 p. m. in the Chinese Room Dr. H. Relton McCarroll will speak on "Congenital Neurofibromatosis" before the Arizona Chapter, Western Orthopedic Association, Wednesday at 12:30 p. m. Dr. John B. Grow will present "Surgery of Pulmonary Suppurative Diseases" before the Arizona Chapter, American College of Chest Physicians. The Arizona Society for Crippled Children will hold an open meeting on hearing conservation at 2 p. m. Wednesday.

CONNECTICUT

Anesthesiologists Meeting.—The annual meeting of the Connecticut State Society of Anesthesiologists will be held in conjunction with the annual meeting of the Connecticut State Medical Society at the Hamden High School in Hamden, April 29. Dr. Vincent J. Collins, director of the department of anesthesiology, St. Vincent's Hospital, New York, will speak on "The Physiology of Position," and Dr. Henry K. Beecher, Dorr professor of research in anesthesia, Harvard Medical School, Boston, on "The Metabolic Effects of Anesthesia."

Meeting on Rheumatism.—The Connecticut Rheumatism Association has scheduled its annual clinical session April 29 at 3:30 p. m. in the Hamden High School in connection with the annual meeting of the Connecticut State Medical Society. Dr. William B. Rawls, chief of the Arthritis Clinic, New York Polyclinic Medical School and Hospital, will speak on "The Intra-Articular Use of Hydrocortisone (Compound F) in Arthritic Joints." His talk will be preceded by a motion picture illustrating the method of administration and the results obtained by this therapy in a large series of clinic patients. Dr. A. Albert LaPlume, Bristol, is in charge of arrangements, and Dr. LeMoyne Copeland Kelly, Waterbury, will preside.

State Medical Meeting in Hamden.—The annual meeting of the Connecticut State Medical Society will be held at Hamden High School April 27-29 under the presidency of Dr. Edward J. Whalen, Hartford. On Wednesday at 2 p. m. there will be a symposium on thyroid disease, arranged by the Connecticut Society of American Board Surgeons, in which the following physicians will participate: Elmer C. Bartels, Boston, George Crile Jr., Cleveland, and Oliver Cope, Boston. Dr. Francis A. Sutherland, Torrington, will preside at a round-table discussion following the symposium. Tuesday afternoon the Connecticut Chapter, American Academy of General Practice, will present discussions on "Cancer in Childhood" by Dr. James P. King, Boston, and "Nutritional Diseases in Infancy with Emphasis on Mucoviscidosis" by Dr. Harry Shwachman, Boston. Other out-of-state speakers include:

Irving S. Wright, New York, Anticoagulant Therapy.
John R. Neefe, Philadelphia, Differential Diagnosis of Jaundice and Application of Liver Function Tests.
Maurice B. Strauss, Framingham, Mass., Drug Treatment in the Anemias.
William T. Green, Boston, Management of Poliomyelitis.
Henry Doublet, New York, Diagnosis and Treatment of Acute and Chronic Pancreatitis.
Henry T. Randall, New York, The Loss and Replacement of Gastrointestinal Fluids.
John I. Brewer, Chicago, Gynecologic and Surgical Aspects of Endometriosis.
Joe V. Meigs, Boston, Treatment of Choice in Carcinoma of the Cervix and Body of the Uterus.

The annual dinner of the society will be held at the Waverly Inn, Cheshire, at 7 p. m. Tuesday. The Woman's Auxiliary to the Connecticut State Medical Society will hold its annual meeting Tuesday at the Woodbridge Country Club.

FLORIDA

State Medical Meeting at Hollywood.—The annual meeting of the Florida Medical Association will be held at the Hollywood Beach Hotel, Hollywood, April 27-29 under the presidency of Dr. Robert B. McIver, Jacksonville. On Tuesday, 11:30 a. m., Dr. Frank H. Lahey, Boston, will speak on surgical lesions of the stomach, duodenum, and jejunum. On Wednesday at 9:30 a. m. Prof. John McMichael, London, England, will speak on hexamethonium therapy in hypertension. Other presentations by out-of-state speakers include:

Pheochromocytoma, M. Eugene Flipse, Miami; discussion, Walter F. Kvale, Rochester, Minn.
Unsolved Problems in Industrial Surgery, Henry C. Marble, Boston.
Cause and Treatment of Nonunion of Tibial Fractures; Indications and Technique for Ankle Fusions, Guy A. Caldwell, New Orleans.
Hematuria, Its Pathological Significance, George F. Cahill, New York.
Surgical Treatment of Cervical and Fundal Malignancies, Joseph H. Pratt, Rochester, Minn.
Therapeutic Research with Radioisotopes, Marshall H. Brucer, Oak Ridge, Tenn.

Monday at 7 p. m. alumni and fraternity suppers will be held as follows: Emory University School of Medicine, Atlanta; Tulane University of Louisiana School of Medicine, New Orleans; Former Mayo Fellows; Duke University School of Medicine, Durham, N. C.; Georgia groups; and Cincinnati groups. At 9 p. m. there will be a smoker (tariff, $2.50 per person). The association dinner ($5.75 per person) will be held Tuesday at 7 p. m. in the Main Dining Room. The Florida Association of Industrial and Railway Surgeons, which will meet Sunday, will hear addresses on hand injuries by Dr.

Physicians are invited to send to this department items of news of general interest, for example, those relating to society activities, new hospitals, education and public health. Programs should be received at least three weeks before the date of meeting.

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Henry C. Marble, Boston, and diagnosis and treatment of low back injuries by Dr. Guy A. Caldwell, New Orleans. At the meeting of the Florida Obstetric and Gynecologic Society Sunday Dr. Joseph H. Pratt, Rochester, Minn., will discuss "Surgical Treatment of Gynecologic Cancer." The afternoon session of the Florida Society of Ophthalmology and Otolaryngology will have as guest speakers Dr. James S. Shipman, Philadelphia ("Detachment of the Retina") and Dr. James A. Harrill, Winston-Salem, N. C. ("Bronchography"). Dr. Clifford G. Grulee Jr., New Orleans, will address the meeting of the Florida Pediatric Society (2 p. m.) on "Sudden and Unexpected Deaths in Infancy," and Dr. Margaret H. D. Smith, New Orleans, at the evening meeting, will discuss "Fulminating Infections, Differential Diagnosis and Management." The program of the Florida Urological Society (Sunday, 2 p. m.) will be presented by Dr. George F. Cahill, New York, who will speak on "Surgical Treatment of Vesical Ectopy and the Handling of Some Complications" and will serve as moderator at a pyelographic clinic.

ILLINOIS

University News.—Dr. David S. Ruhe has been appointed clinical assistant professor of medicine at the University of Illinois, Chicago. He previously was affiliated with Emory University School of Medicine, Emory University, Ga., and New York Medical College, Flower and Fifth Avenue Hospitals.——Dr. Gabriel Langfeldt, professor of psychiatry at the University of Oslo in Norway, who is presently engaged in advanced psychiatric research on a Fulbright fellowship at the Manteno State Hospital, has been appointed lecturer in psychiatry, with the rank of professor, at the University of Illinois College of Medicine.

Chicago

Philosophy of Clinical Trial.—Austin Bradford Hill, Ph.D., director, department of medical statistics and epidemiology, London School of Hygiene and Tropical Medicine, University of London, will lecture on "The Philosophy of the Clinical Trial" April 29 at 5 p. m. at the University of Chicago School of Medicine, 950 E. 59th St., Pathology 117.

Kiwanis Creates Spastic Research Fund.—Problems of the deeper brain areas in cerebral palsy will be studied over the next five years at Children's Memorial Hospital in a project financed by the Illinois-Eastern Iowa District of Kiwanis International, which has reported pledges totaling $82,000 from 159 of its clubs. The team that will carry out the research, all of whom are contributing their services, consists of Drs. Luis V. Amador, John A. Bigler, Joseph D. Boggs, Douglas N. Buchanan, George P. Guibor, John L. Reichert, Charles N. Pease, and Harvey White, all from the Children's Memorial staff. Advisory consultants are Dr. Percival Bailey, Dr. Paul C. Bucy, and Ray Snider, Ph.D. Dr. John Hankinson, London, England, has recently arrived in Chicago as a resident fellow to assist in the work. A special Spastic Paralysis Research Foundation, set up by Kiwanis to administer the research funds, includes the following Illinois physicians: C. Paul White, Kewanee, Ross W. Griswold, Litchfield, George L. Drennan, Jacksonville, Hugh I. Conn, Newman, Frank P. Hammond, and Harry R. Keiser, Chicago. On Jan. 10 Kiwanis leaders and hospital and medical representatives dedicated a bronze plaque at Children's Memorial Hospital to mark the inception of the research project.

IOWA

Personal.—Dr. Frederick F. Null, Hawarden, was recently awarded a plaque by the Business and Professional Woman's Club of Hawarden, in recognition of his "many years of outstanding service." He was described as a man "who stands today as a living embodiment of the rule 'Others first, self second'."——Gov. William S. Beardsley has named the following physicians to the state board of health for terms ending January, 1955: Herbert W. Rathe, Waverly, Herbert E. Stroy, Osceola, Prince E. Sawyer, Sioux City, Milford E. Barnes, Iowa City, and Fred Sternagel, West Des Moines.

State Medical Meeting in Des Moines.—The Iowa State Medical Society will hold its annual meeting April 26-29 at the Hotel Fort Des Moines in Des Moines under the presidency of Dr. Ben T. Whitaker, Boone, who will deliver the presidential address Monday at 9:15 a. m. On Tuesday at 11 a. m. Dr. Louis H. Bauer, Hempstead, N. Y., President of the American Medical Association, will deliver an address. Dr. Bauer will also address the Woman's Auxiliary to the Iowa State Medical Society, meeting concurrently, at luncheon in the Terrace Room of the Hotel Savery the same day. Other out-of-state speakers include:

Leroy A. Calkins, Kansas City, Kan., Occiput Posterior.
Howard K. Gray, Rochester, Minn., Diagnosis and Treatment of Carcinoma of the Stomach with Comments Relating to Encouraging Improvements in the Five Year Survival Rate.
Warren H. Cole, Chicago, Intestinal Obstruction.
Henry T. Ricketts, Chicago, Diabetes Mellitus in General Practice.
Benjamin Felson, Cincinnati, Roentgenology in Obstetrics and Gynecology.
Charles P. Bailey, Philadelphia, Recent Advances in Cardiac Surgery.
James K. Stack, Chicago, Fractures and Casts.
Edward L. Compere, Chicago, Medical Legal Problems in the Handling of Industrial Compensation Cases.
John M. Dorsey, Detroit, Every Man His Own Physician.
J. Robert Wilson, Philadelphia, Pudendal Block Anesthesia in Obstetrics.
Robert H. Monahan, St. Paul, Cause of Senile Cataract.
Clifford F. Lake, Rochester, Minn., Surgical Treatment of Chronic Sinusitis.
Howard B. Hunt, Omaha, Relative Roles of Radiation and Surgery in Treatment of Gynecologic Cancer.

On Tuesday at 4 p. m. at a joint meeting of the medical and pediatric sections, a symposium on poliomyelitis will be held, Dr. Abe B. Baker, Minneapolis, presenting "Bulbar and Respiratory Poliomyelitis," and Dr. Miland E. Knapp, Minneapolis, "Rehabilitation of Severe Poliomyelitis Paralytics." The annual banquet at 7 p. m. Tuesday will be preceded by a social hour in the Grand Ballroom of the Hotel Fort Des Moines. The annual banquet of the American Medical Women's Association, which will have as guest speaker Dr. Edith L. Potter, Chicago, will be held Monday at 6:30 p. m. Special luncheons and dinners have been arranged by various organizations, including the Iowa Academy of General Practice, Iowa Association of Pathologists, Iowa Society of Anesthesiologists, Iowa X-Ray Club, Iowa Pediatric Society, and the Iowa Neuropsychiatric Society. The annual golf tournament of the association will start Sunday at 10 a. m. at the Golf and Country Club.

KENTUCKY

Community Honors Dr. Collins.—Dr. Lister Collins, Mount Eden, was recently honored for his 50 years of medical service in Spencer and Anderson counties at a dinner given by his community. A television set was presented to Dr. Collins at the ceremonies, which were sponsored by the Mount Eden Baptist and Christian Churches on the occasion of his 76th birthday.

MARYLAND

State Medical Meeting in Baltimore.—The Medical and Chirurgical Faculty of the State of Maryland will hold its annual meeting at 1211 Cathedral St., Baltimore, April 28-29, under the presidency of Dr. Maurice C. Pincoffs, Baltimore, who will give the presidential address on "Thoughts Concerning the Future of Medicine" Tuesday at 8 p. m. in Osler Hall. The annual ball will be April 27 at the Alcazar, Cathedral and Madison Streets, under the sponsorship of the Woman's Auxiliary to the Baltimore City Medical Society. Dr. Louis H. Bauer, Hempstead, N. Y., President of the American Medical Association, will be guest speaker at the Woman's Auxiliary luncheon in the Ballroom of the Sheraton Belvedere Hotel Wednesday, 12:30 p. m., and Wednesday evening he will be the guest at the state meeting in Osler Hall, 8 p. m. Other out-of-state speakers on Tuesday include Dr. Robert P. Glover, Philadelphia, on "Indications and Results in Cardiac Surgery," 11 a. m.; Dr. Thaddeus S. Danowski, Pittsburgh, who will deliver the Harvey Grant Beck memorial lectureship on "Interrelations of Thyroid and Iodine Metabolism," 3:10 p. m., and Dr. Robert R. Linton, Brookline, Mass., who will give the John M. T. Finney Fund lecture on "Surgical Treatment of Bleeding Esophageal Varices," 8 p. m. On Wednesday at

4:10 p. m., Dr. Frank G. MacMurray, Washington, D. C., will discuss "Cat Scratch Disease" and at 8 p. m. Dr. John S. L. Browne, Montreal, Canada, will present the I. Ridgeway Trimble Fund lecture on "The Changing Nature of Medicine."

MASSACHUSETTS

Alpha Omega Alpha Lecture.—The Boston University Chapter of Alpha Omega Alpha will sponsor a lecture on "Recent Advances of Purine Metabolism and Their Relationship to the Pathogenesis of Gout" by Dr. Alexander B. Gutman, Columbia University College of Physicians and Surgeons, New York, April 27, 5 p. m. in the Boston University School of Medicine auditorium, 80 E. Concord St. The address is open to the medical profession.

New England Center Lectures.—The House Officers' Association of New England Center Hospital, Boston, announces that at 7 p. m. May 1 Dr. Garfield G. Duncan, clinical professor of medicine, Jefferson Medical College of Philadelphia, will speak on "Practical Considerations in the Management of Diabetes." Discussors will be Drs. Priscilla White and Joseph Rosenthal, Boston. The program will be held in the Stearns Auditorium of the hospital. All interested persons are invited.

MINNESOTA

University News.—The first in a series of "Family Doctors' Days" will be presented at the University of Minnesota Hospitals, Minneapolis, April 29. The following program, to which all physicians are invited, will be presented by the department of medicine:

12 noon, Luncheon with department of medicine staff.
2 p.m., Antibiotics in Medical Practice, Wesley W. Spink.
3 p.m., Ward Rounds, Cecil J. Watson.
4:30 p.m., Clinicopathological Conference, Cecil J. Watson and staff.

Jackson Lecture.—The annual Clarence M. Jackson lecture on "Gastrointestinal Symptoms with Particular Reference to Motor Disturbances" will be presented on the University of Minnesota campus, Minneapolis, at 8 p. m., April 28, by Dr. Chester M. Jones, clinical professor of medicine, Harvard Medical School, Boston. Dr. Jones will also participate in a continuation course in gastroenterology for general physicians, which will be held at the Center for Continuation Study April 27-29.

MISSOURI

Meeting on Tuberculosis.—The Missouri Chapter of the American College of Chest Physicians and the Missouri Trudeau Society will hold a combined luncheon meeting and x-ray conference in the Hotel President, Kansas City, April 26, when the following program will be presented:

Chemotherapy of Tuberculous Meningitis, Karl H. Pfuetze, Chicago.
Prevention of Postoperative Bronchopulmonary Complications, David B. Radner, Chicago.
Surgical Treatment of Lung Abscess, John H. Mayer, Kansas City.

Dr. Lawrence E. Wood, Kansas City, will serve as moderator for the x-ray conference.

Annual State Medical Meeting.—The annual session of the Missouri State Medical Association will be held in the Municipal Auditorium, Kansas City, April 26-29, under the presidency of Dr. J. William Thompson, St. Louis. Out-of-state speakers include:

Karl H. Pfuetze, Chicago, Newer Chemotherapy of Tuberculosis.
Willis E. Brown, Little Rock, Ark., Management of Eclampsia: A Clinical Evaluation of the Several Therapeutic Schedules.
Darrell A. Campbell, Ann Arbor, Mich., Management of Chronic Obstruction to the Lymphatic and Venous Channels.
Cotter Hirschberg, Topeka, Kan., Emotional Problems of Childhood: Their Management in General Practice.
W. Clark Wescoe, Kansas City, Kan., Responsibilities of the Medical School to the State It Serves.
Douglas W. Macomber, Denver, Immediate Care of Facial Injuries.
Arnold S. Jackson, Madison, Wis., Present Concepts in Treatment of Hyperthyroidism.
Sydney S. Gellis, Boston, Pediatric Emergencies in the Newborn.
Herbert C. Miller, Kansas City, Kan., Pediatric Emergencies in the Older Child.
Ralph A. Dorner, Des Moines, Iowa, Chest Injuries: Diagnosis and Treatment.
Charles M. Blackburn, Rochester, Minn., Medical Aspects of Thyroid Disease.

Daily round-table luncheons will be held, and motion pictures will precede morning and afternoon sessions, with three dimensional slides being shown on Wednesday at 8 a. m. in the Little Theater of the auditorium. Wednesday at 11 a. m. there will be a panel discussion on trauma with the following participants: Dr. Gene B. Starkloff, St. Louis (abdominal injuries); Dr. William J. Stewart, Columbia (orthopedic injuries); and Dr. James C. Cope, Columbia (genitourinary injuries).

NEW YORK

Two Scholarships for Trudeau School of Tuberculosis.—Brooklyn physicians are offered two four-week scholarships, including tuition, Pullman transportation, and $200 for room and board at the Trudeau School of Tuberculosis, Saranac Lake, June 1-27, by the Brooklyn Tuberculosis and Health Association. Application forms and copy of the schedule of the course may be secured from the Brooklyn Tuberculosis and Health Association, 285-293 Schermerhorn St., Room 303, phone MAin 4-8531. Applications must be received by May 1.

Hospital News.—The Convalescent Hospital for Children, Rochester, recently received a bequest of $18,742 under the will of the late Mrs. Frances R. Heughes.——The Division of Cardiology, Department of Medicine, Jewish Hospital of Brooklyn, will present a lecture on "Advances in Clinical Cardiology" by Dr. Charles K. Friedberg, Mount Sinai Hospital, New York, April 30 at the Louria Auditorium, 555 Prospect Place. On May 14 in the same auditorium "Advances in Cardiovascular Surgery" will be discussed by Dr. Ralph A. Deterling Jr. of Columbia University College of Physicians and Surgeons, New York.

New York City

Lecture on Leukemia.—On May 1, 3 p. m., the division of neoplastic diseases, Montefiore Hospital for Chronic Diseases, will present "The Balance Concept of Leukemia in Man" by Dr. Howard R. Bierman, San Francisco.

Lectures for General Practitioners.—In its Saturday morning seminar the Department of Health of the City of New York will present the following lectures in May:

May 2, The Doctor's Office—A Cancer Detection Center, Abraham Oppenheim.
May 9, Newer Aspects of Thyroid Disease, Sidney C. Werner.
May 16, Clinical Observations on Migraine and Its Treatment, Solomon Hirsch.

Lecture on Oral Pathology.—Dr. Oscar V. Batson, Medico-Chirurgical College, Graduate School of Medicine, University of Pennsylvania, Philadelphia, will read a paper on "The Venous Networks About the Mouth and Jaws" at the monthly conference of the New York Institute of Clinical Oral Pathology, 9 p. m. April 27 in room 440, New York Academy of Medicine.

Aaron Brown Lecture.—The Cornell University Medical College chapter of Phi Delta Epsilon will present its annual Aaron Brown memorial lecture May 1, 8 p. m., in the college auditorium, 1300 York Ave. Dr. Charles P. Bailey, professor of surgery, Hahnemann Medical College and Hospital of Philadelphia, will speak on "The Development and Future of Cardiac Surgery." Physicians and students are invited.

Pediatric Course in Malignancy.—The pediatric department of the Memorial Center for Cancer and Allied Diseases (444 E. 68th St.) announces a course April 29 to May 1 for pediatricians, general practitioners, and health officers. Current developments and established methods in diagnosis, differential diagnosis, and management of benign and malignant tumors, Hodgkin's disease, leukemia, and reticuloendotheliose in childhood are included. There will be demonstrations, examinations of children, and ward rounds and seminars under the direction of the staffs of the Memorial Center and Sloan-Kettering Institute for Cancer Research. Fee, $35.

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OREGON

Personal.—Dr. Sam B. Liu, Chinese-American physician, was recently honored as the "citizen of the month" at a luncheon meeting of the Portland Americanization Council, at which time a certificate of citizenship was presented to him.

Society News.—The Alumni Association of the University of Oregon Medical School held its annual meeting at the school April 22-24 jointly with the Sommer memorial lectures and the annual meeting of the Oregon Chapter, American Academy of General Practice. The Sommer memorial lecturers and their presentations were as follows:

Thomas M. Durant, Philadelphia, (1) Congestive Failure: Pathophysiology and Management of Resistant Cases; (2) Cardiac Arrhythmias: Recognition and Management; (3) Coronary Disease: Etiology and Management.

W. Edward Chamberlain, Philadelphia, (1) What the General Practitioner Should Know About the Biological Effects of Irradiation; (2) Low Back Pain; (3) General Considerations in the Treatment of Cancer.

Frederic H. Bentley, Newcastle-on-the-Tyne, England, (1) Surgery of the First Part of Duodenum; (2) Management of Chronic Vascular Occlusion.

Dr. Bentley served as moderator for the Sommer lecture panel on Friday.

PENNSYLVANIA

Postgraduate Institute.—The Philadelphia County Medical Society will hold its postgraduate institute April 28 to May 1 at the Bellevue-Stratford Hotel, Philadelphia. On Tuesday morning there will be a panel discussion on "Newer Drugs and Their Place in Therapy." A symposium on "Toxemia of Pregnancy" Tuesday afternoon will be preceded by a panel discussion on "Newer Approaches to Older Patients," with Albert I. Lansing, Ph.D., St. Louis, as guest speaker. TV programs from Philadelphia General Hospital will be given Wednesday and Thursday mornings. A symposium on pediatrics will follow the Wednesday afternoon panel discussion on "Endocrinology," and a panel discussion on "Heart Surgery: Indication and Results" Thursday afternoon will precede a symposium on "What Surgery Has to Offer," which will include discussions on bleeding ulcer, hypertension, chronic pancreatitis, and bronchiectasis. A clinicopathological conference will be held in the evening. A symposium on gynecology and a tumor conference are scheduled for Friday morning, and a panel discussion on "Liver Disease" Friday afternoon.

Pittsburgh

Theobald Smith Award.—Dr. Frank J. Dixon Jr., chairman of the department of pathology, University of Pittsburgh School of Medicine, has been named winner of the 1952 Theobald Smith Award in Medical Sciences for his work on tracer methods in approaching antigen-antibody problems. The award, which is limited to researchers under 35, was established by Eli Lilly and Company in 1936 and is administered by the American Association for the Advancement of Science. Dr. Dixon formerly served as a research assistant at Harvard University, Boston, and as instructor in pathology at Washington University, St. Louis.

TEXAS

Dr. Pullen Goes to Missouri.—Dr. Roscoe L. Pullen, dean of the University of Texas Postgraduate School of Medicine, Houston, was recently appointed dean of the University of Missouri School of Medicine, Columbia; professor of medicine; director of the University Hospitals; and director and consultant of the Missouri State Crippled Children's Service.

Visiting Scientists.—Walter Hild of Hamburg, Germany, a Rockefeller Foundation fellow, is visiting the University of Texas Medical Branch, Galveston, for several months for a special study in the tissue culture laboratory under the direction of Charles M. Pomerat, Ph.D. He will investigate the neurosecretory functions of the anterior pituitary.——Werner Kunast of Hamburg, Germany, has been appointed teaching and research fellow in radiology at the University of Texas Medical Branch, Galveston.

Centennial Session of State Society at Houston.—The centennial anniversary session of the Texas Medical Association will be held at the Shamrock Hotel, Houston, April 26-29, under the presidency of Dr. Truman C. Terrell, Fort Worth, who will deliver the presidential address Tuesday, 10:40 a. m. The president's banquet will be held Tuesday, 8 to 10 p. m. Dr. Edward J. McCormick, Toledo, Ohio, President-Elect of the American Medical Association, will deliver an address on "Medical Progress and the American Medical Association." Other out-of-state speakers include:

Francis F. Rosenbaum, Milwaukee, Recognition and Management of Paroxysmal Rapid Heart Action.

Reuben B. Chrisman Jr., Miami, Fla., The Physician, His Wife, and Their Relation to the Government as Citizens.

Elmer Hess, Erie, Pa., The Patient and the Proper Function of the Doctor's Office.

Mr. Rollen W. Waterson, Oakland, Calif., Public Relation Responsibilities of the County Medical Society and Auxiliary.

A feature of the celebration will be publication of "A History of the Texas Medical Association 1853-1953" by Dr. Pat I. Nixon, San Antonio, Texas. The Texas Air-Medics Association and the Texas Dermatological Association have scheduled meetings for Sunday and Monday. The Texas Chapter, American College of Chest Physicians, will have a luncheon panel discussion Monday on bronchiectasis and at 4 p. m. an x-ray conference. The Texas Diabetes Association will present a panel discussion on "Problems in the Treatment of Diabetes" at 11 a. m. Sunday. Dr. George E. Burch, New Orleans, will address the Texas Heart Association Monday, 3:45 p. m., on "Management of Angina Pectoris," and Dr. Stewart G. Wolf Jr., Oklahoma City, will present "Practical Considerations in the Physiology of Pain with Special Reference to Treatment" before the meeting of the Texas Neuropsychiatric Association Monday, 11 a. m., and "Physiologic Effects of Placebo Administration" at 3:30 p. m. The Texas Orthopedic Association, Texas Railway and Traumatic Surgical Association, and Texas Society of Anesthesiologists will meet on Monday. At the meeting of the Texas Society of Gastroenterologists and Proctologists, Monday, 2 p. m., Dr. James E. Thompson, New York, will present "Experience with Carcinoma of the Colon."

UTAH

University News.—The Ford Foundation and the government of India have requested the services of Dr. John Z. Bowers, dean, University of Utah College of Medicine, Salt Lake City, to organize public health training based on recommendations that he made after a mission to India last summer. He left for India March 15 for two months.——Dr. Richard H. Young, former dean at the University of Utah College of Medicine, presently dean of Northwestern University Medical School, Chicago, was guest speaker at the annual medical college alumni banquet at the University of Utah, March 12.

VERMONT

Phi Delta Epsilon Lecture.—The annual Phi Delta Epsilon lecture at the University of Vermont, Burlington, was given April 17 by Homer W. Smith, Sc.D., professor of physiology, New York University College of Medicine, on "The Development of Modern Renal Physiology."

Annual Cancer Symposium.—The Vermont State Medical Society announces that the annual cancer symposium will be held at the University of Vermont, Burlington, April 29. The sessions will open with the showing of a film on uterine cancer. Panel discussions will be presented on diagnosis and treatment of uterine cancer and the role of the general practitioner, diagnosis and treatment of gastrointestinal cancer and the role of the general practitioner, breast cancer and the role of the general practitioner, and cancer of the lung, head, and neck and the role of the general practitioner. Moderators will be Drs. Ernest M. Daland, Walpole, Mass., and William G. Cahan, New York. The dinner meeting at 7 p. m. will be addressed by Dr. George A. Wolf Jr., dean, University of Vermont College of Medicine, who will speak on "The Role of the University of Vermont, College of Medicine, in the Problem of Cancer in Vermont."

VIRGINIA

Annual Clinic Day in Alexandria.—The annual Clinic Day sponsored by the Alexandria Medical Society will be held April 26 at the George Mason Hotel, Alexandria. The fee of $2 includes lunch and the social hour (5 to 7 p. m.) at the conclusion of the following scientific program, which will be presented by the faculty of the Johns Hopkins University School of Medicine, Baltimore:

Current Concepts of the Collagen Diseases, Morgan Berthrong.
Evaluation of the Adult for Cardiac Surgery, Edwin Cowles Andrus.
Developments in the Recognition and Treatment of Hypersplenism, Carroll Lockard Conley.
Judgment in Gynecologic Surgery, Emil Novak.
Fluids and Electrolyte Balance in Children, Harold E. Harrison.
Seminar on the Emotional Aspects of Hypertension, Caroline C. Bedell Thomas.

WASHINGTON

Society News.—The annual meeting of the Spokane Society of Internal Medicine will be held April 25 at the Ridpath Hotel, Spokane. Guest speakers, Drs. Howard P. Lewis, Daniel H. Labby, University of Oregon Medical School, Portland, and Dr. Wade Volwiler, University of Washington School of Medicine, Seattle, will conduct a clinical conference on unusual cases of liver disease in the Providence Auditorium of Sacred Heart Hospital, Spokane, 9:15 to 11:15 a. m., and a symposium on liver disease at the Ridpath Hotel, at 1:30 p. m. The informal banquet, 7:30 p. m., will be preceded by a social hour and will be addressed by Dr. Lewis on "Thoughts on the Selection and Training of Students of Medicine."

WEST VIRGINIA

Film Reading Session.—The West Virginia State Medical Association, which will hold its annual meeting at White Sulphur Springs, July 23-25, invites members to submit films of interesting cases, with a short history, for possible presentation during an x-ray clinic or film reading session by a panel of outstanding roentgenologists. Cases must be diagnosed and the diagnosis confirmed by pathological reports following operation, or postmortem examination. Material should be mailed to the moderator of the clinic, Dr. Vernon L. Peterson, 310 Medical Arts Bldg., Charleston.

GENERAL

Cleft Palate Rehabilitation.—The American Association for Cleft Palate Rehabilitation will hold its annual meeting at the Atlanta-Biltmore Hotel in Atlanta April 27-28. The program will include section meetings, motion pictures, and special exhibits. The meeting will be open to all persons interested in cleft palate rehabilitation. Information may be obtained from Willard T. Hunnicutt, D.D.S., 302 Medical Arts Building, Atlanta.

Meeting of Laryngologists.—The American Laryngological Association will hold its annual meeting at the Roosevelt Hotel, New Orleans, April 26-27 under the presidency of Dr. Louis H. Clerf, Philadelphia. Speakers by invitation include Drs. A. S. MacMillan, Boston, John J. O'Keefe, Philadelphia, H. Russell Fisher, Los Angeles, and John Adriani, New Orleans. The president's reception to fellows and their ladies will be Sunday at 6:30 p. m.

Gastroenterologists Meet in Atlantic City.—The annual meeting of the American Gastroenterological Association will be held at the Hotel Claridge in Atlantic City, N. J., May 1-2, under the presidency of Dr. Albert M. Snell, Palo Alto, Calif., whose address will be delivered Saturday morning. More than 30 papers will be presented. Friday afternoon Dr. Hugh R. Butt, Rochester, Minn., will serve as moderator for a panel on the clinical and biochemical features of hepatic insufficiency.

Lasker Public Health Awards.—One of the 1952 Lasker awards was recently bestowed by the American Public Health Association on Dr. Howard A. Rusk, director of the Institute of Physical Medicine and Rehabilitation of the New York University-Bellevue Medical Center, for "distinguished services to humanity through rehabilitation." He was also honored for "achievement in the development of professional training for rehabilitation and for education of the public in the problems of administration of health and medical service."

Otologists Meet in New Orleans.—The annual meeting of the American Otological Society, Inc., will be held May 1 and 2 at the Roosevelt Hotel, New Orleans, under the presidency of Dr. Albert C. Furstenberg, Ann Arbor, Mich. Presentations by invitation include:

Heinrich F. G. Kobrak, Chicago, Experimental Observations on Sound Conduction in the Middle and Inner Ear.
Anderson C. Hilding, Duluth, Minn., Theoretical Considerations of the Transmission of Sound Vibrations from the Perilymph to the Organ of Corti.
John H. T. Rambo and Dorothy Wolff, Ph.D., New York, An Experimental Study of the Influence of the Autonomic Nervous System on the Inner Ear.

American Academy of Child Psychiatry.—This group, organized in Cleveland, Feb. 22, elected as its officers the following physicians: president, George E. Gardner, Boston; president-elect, Frederick H. Allen, Philadelphia; secretary, Frank J. Curran, Charlottesville, Va.; and treasurer, Mabel Ross, New York. Membership in the academy is limited to physicians whose chief professional interest and activity are in the field of child psychiatry, who are members of the American Psychiatric Association, and who have had at least two years' training in a clinic setting deemed adequate to give training in child psychiatry and five years' experience in child psychiatry after completion of the training period. The academy is planning a scientific program in Los Angeles, May 4.

Industrial Medical Practice.—Under the joint sponsorship of the Harvard University School of Public Health, the American Academy of General Practice, the A. M. A. Council on Industrial Health, and the Liberty Mutual Insurance Companies, a seminar on the fundamentals of industrial medicine was held April 3-4 at the school (55 Shattuck St., Boston). Subjects for discussion were industrial injuries, with emphasis on compensation laws, claims handling, and safety; occupational diseases, with particular attention to problems of diagnosis and of evaluation of the severity of a toxic exposure; industrial medical practice, including its aims and scope, its industrial relations aspects, and its relationship to private practice; and the role of industrial medicine in the country's problems of medical care.

National Academy of Sciences.—The annual meeting of the National Academy of Sciences will be held April 27-29 at the academy headquarters, 2101 Constitution Ave., Washington, D. C. Scientific sessions are scheduled for Monday and Wednesday, morning sessions beginning at 10 o'clock and the afternoon sessions at 2. At a public meeting Monday, 8 p. m., presentations will be made of the Agassiz, Elliot, Kovalenko, Thompson, and Walcott medals. Admission for members and their guests will be by ticket, which may be secured at the registration desk. On Tuesday the academy dinner will be held at the Hotel Washington, 15th Street and Pennsylvania Avenue, N.W. Members may bring their wives and other guests. Wednesday evening there will be a buffet supper for members only.

Meeting on Neoplastic Diseases.—The American Association for the Study of Neoplastic Diseases will hold its annual meeting at St. Agnes Hospital, Baltimore, April 30 to May 2 under the presidency of Dr. Eugene R. Whitmore, Washington, D. C. Thursday at 8 p. m. Dr. George T. Pack, New York, will present "Pigmented Nevi and Melanomas." Dr. Elmer Hess, Erie, Pa., the guest speaker Friday morning, will discuss "Lesions of the Male Genitourinary System." Among guest speakers who will talk on "Radioisotopes and Their Use in Diagnosis and Treatment" Friday afternoon are Drs. Samuel P. Asper and Morgan Berthrong, Johns Hopkins Hospital, Baltimore. Friday evening Dr. Elbert DeCoursey, Washington, D. C., will address the dinner meeting, 7 p. m., at the Carnical Room, Lord Baltimore Hotel. Saturday morning will be devoted to a diagnostic session on radiology, for which Dr. Ramsey Spillman, New York, will serve as chairman.

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Annual Symposium on Venereal Diseases.—The annual symposium on "Recent Advances in the Study of the Venereal Diseases" will be held April 30 to May 1 in Washington, D. C., under the auspices of the Experimental Therapeutics Study Section, National Institutes of Health, U. S. Public Health Service, and in conjunction with the 15th annual session of the American Venereal Disease Association. The meetings will be held in the auditorium of the Federal Security Building, Independence Avenue between Third and Fourth Streets, S.W., where Dr. Leonard A. Scheele, Washington, D. C., surgeon general of the U. S. Public Health Service, will give the address of welcome Thursday at 10 a. m. Dr. John J. Wright, Chapel Hill, N. C., will deliver the presidential address before the American Venereal Disease Association at 10 a. m. Friday. Thirty-five presentations have been scheduled, and 11 papers will be read by title. The U. S. Navy film "Stopping the Spread" will be shown at the end of the Thursday afternoon session.

Meeting of Maxillofacial Surgeons.—The annual meeting of the American Society of Maxillofacial Surgeons will be held at the Wade Park Manor Hotel, Cleveland, April 26-29 under the presidency of Dr. John W. Gerrie, Montreal, Canada. The Monday morning and Tuesday afternoon sessions will be held in the fifth floor amphitheater, University Hospitals. At 1:30 p. m. Drs. Claude S. Beck and David S. Leighninger, Cleveland, will present "Revascularization of the Heart for Coronary Artery Disease," and from 2 to 5 p. m. "Resuscitation of the Heart" (motion picture and laboratory demonstration) will be presented by Drs. Beck, Leighninger, and Robert M. Hosler. Single presentations by invitation include:

William D. Holden, Cleveland, Use of Isotopes in Surgical Research.
Stephen Hudack, Cleveland, Healing in Maxillofacial Structures and Membrane Bones.
Donald M. Glover, Cleveland, Differential Diagnosis of Neck Swelling.
Orion H. Stuteville, Evanston, Ill., Causes of Failure in Cleft Palate Surgery.
H. Hoyle Campbell, Toronto, Canada, Surgery of Lesions of the Upper Face.
Adolph M. Brown, Chicago, Prosthetic Corrections of Inoperable Deformities of the Face.

Meeting of Otorhinolaryngologists.—The American Laryngological, Rhinological and Otological Society, Inc., will hold its annual meeting at the Hotel Roosevelt, New Orleans, April 28-30, under the presidency of Dr. Francis E. LeJeune, New Orleans, who will introduce the guest of honor, Dr. Charles A. Thigpen, Montgomery, Ala. Dr. Louis H. Bauer, Hempstead, N. Y., President of the American Medical Association, will address the meeting Thursday, 10:15 a. m. Other speakers by invitation include:

Harold F. Schuknecht and Richard Woellner, B.A., Chicago, Low Tone Deafness from Eighth Nerve Section (with slides).
Alton Ochsner, New Orleans, Bronchogenic Carcinoma: The Role of the Bronchoscopist in Its Early Diagnosis.
Gordon H. Hoople and Louis M. DiCarlo, Ph.D., Syracuse, N. Y., The Formation of a Hearing and Speech Center.
Maurice H. Cottle, Chicago, The Role of Rhinoplasty in Rhinology.
Frederick A. Figi and David B. Stark, Rochester, Minn., Neurofibromas of the Larynx—Report of Cases: Motion Pictures.
Louis H. Clerf and William H. Baltzell, Philadelphia, A Reevaluation of Semon's Hypothesis.

The society and the American Broncho-Esophagological Association will be joint hosts at a sazerac party Wednesday, 6-7 p. m. in the Grand Ballroom.

Allergists Meeting in Chicago.—The annual congress of the American College of Allergists, Inc., will be held at the Hotel Conrad Hilton, Chicago, April 27-29, under the presidency of Dr. J. Warrick Thomas, Richmond, Va., who will deliver the presidential address at 9 a. m. Tuesday. Monday there will be a round-table discussion on dermatological allergy at 7:30 p. m. This will be followed by a clinical panel on psychosomatic allergy, with Dr. Hyman Miller, Beverly Hills, Calif., as moderator. Findings from four seminars on "Emotional Factors in Allergic Disorders," which will conclude the graduate instructional course preceding the annual congress, will be summarized during this session, and trends in the practice of allergy as indicated by answers to the questionnaire "How Do You Feel About Feelings?" will be summarized by Dr. M. Murray Peshkin, New York, president-elect of the college. Charles M. Pomerat, Ph.D., professor of cytology, University of Texas Medical Branch, Galveston, guest speaker of the congress, will present "Direct Observations on Human Allergic Cells with Tissue Culture Technique" Tuesday, 9:25 a. m. A round-table luncheon on pediatric allergy will be held at 12:30 p. m. Tuesday. Dr. Jerome Glaser, Rochester, N. Y., will serve as moderator, with the following participants by invitation: Drs. Arthur H. Rosenblum, Ralph H. Kunstadter, and Ralph Spaeth, Chicago, and Dr. Maximilian Berkowitz, Haifa, Israel (visiting fellow in pediatric allergy, Clinic of Dr. Jerome Glaser, Rochester, N. Y.). Other presentations by invitation include:

Microscopic Observations of the Intrahepatic Circulation of Living Guinea Pigs Before and During Anaphylaxis, Walter S. Burrage, Boston.
Bronchial Insufficiency, Edwin R. Levine, Chicago.
Preliminary Study of Radioactive Ragweed Pollen, George V. LeRoy, Chicago.
Allergic Dermatoses in Industry, Raymond R. Suskind, Cincinnati.
General Management of Dermatitis, Robert R. Kierland, Rochester, Minn.
Relationship of Foods to Asthma, Harold I. Lecks, Philadelphia.
Emotional Aspects of Pediatric Allergy, Johann R. Marx, Denver.

The banquet will be held Tuesday at 7 p. m. in the Grand Ballroom.

EXAMINATIONS AND LICENSURE

NATIONAL BOARD OF MEDICAL EXAMINERS

NATIONAL BOARD OF MEDICAL EXAMINERS: *Parts I and II.* All centers where there are five or more candidates, June 22-24 and Sept. 8-10 (Part I only). Candidates may file applications at any time, but the National Board must receive them at least six weeks before the date of the examination they wish to take. *Part III.* June 9-26. Examination will be held in each of 31 centers having five or more eligible candidates. Exec. Sec., Dr. John P. Hubbard, 133 South 36th St., Philadelphia 4.

EXAMINING BOARDS IN SPECIALTIES

AMERICAN BOARD OF ANESTHESIOLOGY: *Written.* Various locations, July 17. Final date for filing applications was January 17. Sec., Dr. C. B. Hickcox, 80 Seymour St., Hartford 15.

AMERICAN BOARD OF DERMATOLOGY AND SYPHILOLOGY: All candidates must now pass a written examination. *Written.* Various centers, Sept. 3. *Oral.* Philadelphia, Oct. 16-18. To be eligible, candidate must complete 36 months of training before Oct. 1. Final date for filing applications is May 1. Exec. Sec., Miss Janet Newkirk, 66 East 66th St., New York 21.

AMERICAN BOARD OF INTERNAL MEDICINE: *Oral.* New York City, May. The closing date for acceptance of applications for oral examinations was January 2 except for candidates in military or Naval Service. *Oral.* San Francisco, Sept. 21-23, Chicago, Nov. 30-Dec. 2. The closing date for acceptance of applications for the San Francisco and Chicago oral examination was April 1. *Oral Examinations in the Subspecialties.* Allergy, New York City, June. Cardiovascular Disease, New York City, May 27, and Los Angeles, May. The closing date for acceptance of applications was February 1. *Written.* October 19. The closing date for acceptance of applications is May 1. Exec. Sec.-Treas., Dr. William A. Werrell, 1 West Main St., Madison 3.

AMERICAN BOARD OF NEUROLOGICAL SURGERY: *Oral.* Chicago, May or June 1953. Final date for filing application for the oral examination was Jan. 15, 1953. Sec., Dr. Leonard T. Furlow, Washington University School of Medicine, Kingshighway and Euclid Ave., St. Louis.

AMERICAN BOARD OF OBSTETRICS AND GYNECOLOGY: *Oral and Pathological, Part II.* Chicago, May 17-24. Final date for filing application was Feb. 1. Sec., Dr. R. L. Faulkner, 2105 Adelbert Road, Cleveland 6.

AMERICAN BOARD OF OPHTHALMOLOGY: *Practical.* New York City, June 6-10; Chicago, Oct. 5-9. *Written.* Various Centers, January 1954. Final date for filing applications is July 1. Sec., Dr. Edwin B. Dunphy, 56 Ivie Road, Cape Cottage, Maine.

AMERICAN BOARD OF OTOLARYNGOLOGY: *Oral.* Chicago, Oct. 5-9. Sec., Dr. Dean M. Lierle, University Hospital, Iowa City.

AMERICAN BOARD OF PEDIATRICS: *Oral.* Philadelphia, May 1-3; Ann Arbor, June 26-27. Place undecided, Oct. 9-11 (tentative); Indianapolis, November. *Oral.* Pediatric Allergy. Chicago, April 28. Exec. Sec., Dr. John McK. Mitchell, 6 Cushman Road, Rosemont, Pa.

AMERICAN BOARD OF PHYSICAL MEDICINE AND REHABILITATION. *Oral and Written.* May 30-31. Final date for filing applications was March 31. Sec., Dr. Robert L. Bennett, 30 N. Michigan Blvd., Chicago.

AMERICAN BOARD OF PLASTIC SURGERY: New Orleans, May 9-11. Final date for receipt of case reports for the spring examination was January 1. Final date for receipt of case reports for the fall examination (October-November) is June 1 of each year. Corres. Sec., Mrs. Estelle E. Hillerich, 4647 Pershing Ave., St. Louis 8, Mo.

AMERICAN BOARD OF PREVENTIVE MEDICINE: April 23-25, Berkeley, Boston, Baltimore, Minneapolis and New Orleans. New York City, Nov. 7-9. Sec., Dr. Ernest L. Stebbins, 615 North Wolfe St., Baltimore 5.

AMERICAN BOARD OF PROCTOLOGY: *Part I.* For candidates in proctology and ano-rectal surgery. May 9, Kansas City, Minneapolis, Philadelphia and San Francisco. The examination will be in anatomy, physiology, biochemistry and pathology and will be both oral and written. Sec., Dr. Louis A. Buie, 102-110 Second Ave., S.W., Rochester, Minn.

AMERICAN BOARD OF PSYCHIATRY AND NEUROLOGY: San Francisco, April 30-May 1. Sec.-Treas., Dr. David A. Boyd, Jr., 102-110 Second Ave., S.W., Rochester, Minn.

MEETINGS

AMERICAN MEDICAL ASSOCIATION: Dr. George F. Lull, 535 North Dearborn St., Chicago 10, Secretary.

1953 Annual Session, New York, June 1-5.

1953 Clinical Session, St. Louis, Dec. 1-4.

1954 Annual Session, San Francisco, June 21-25.

1954 Clinical Session, Miami, Florida, Nov. 30-Dec. 3.

AERO MEDICAL ASSOCIATION, Biltmore Hotel, Los Angeles, May 11-13. Dr. Thomas H. Sutherland, P. O. Box 26, Marion, Ohio, Secretary.

AMERICAN ACADEMY OF TUBERCULOSIS PHYSICIANS, Hotel Roosevelt, New York, May 30. Dr. Oscar S. Levin, P. O. Box 7011, Denver 6, Secretary.

AMERICAN ASSOCIATION FOR CLEFT PALATE REHABILITATION, The Atlanta-Biltmore, Atlanta, Ga., April 27-28. Dr. Jack Matthews, 1617 Cathedral of Learning, University of Pittsburgh, Pittsburgh 13, Secretary.

AMERICAN ASSOCIATION OF GENITO-URINARY SURGEONS, The Homestead, Hot Springs, Va., May 6-8. Dr. Norris J. Heckel, 122 S. Michigan Blvd., Chicago 3, Secretary.

AMERICAN ASSOCIATION ON MENTAL DEFICIENCY, Hotel Statler, Los Angeles, May 12-16. Dr. Neil A. Dayton, P. O. Box 96, Willimantic, Conn., Secretary.

AMERICAN ASSOCIATION FOR THE STUDY OF NEOPLASTIC DISEASES, Lord Baltimore Hotel, Baltimore, April 30-May 2. Dr. Bruce H. Sisler, Gatlinburg, Tenn., Secretary.

AMERICAN BLOOD IRRADIATION SOCIETY, Waldorf-Astoria, New York, May 30-31. Dr. H. T. Lewis, 1241 Peermont Ave., Pittsburgh, Secretary.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION, Roosevelt Hotel, New Orleans, April 28-29. Dr. Edwin N. Broyles, 1100 North Charles St., Baltimore 1, Secretary.

AMERICAN COLLEGE OF ALLERGISTS, Conrad Hilton Hotel, Chicago, April 26-29. Dr. Fred W. Wittich, 423 LaSalle Medical Bldg., Minneapolis 2, Secretary.

AMERICAN COLLEGE OF CARDIOLOGY, Hotel Statler, Washington, D. C., June 7-9. Dr. Philip Reichert, 480 Park Ave., New York 22, Secretary.

AMERICAN COLLEGE OF CHEST PHYSICIANS, Hotel New Yorker, New York, May 28-31. Mr. Murray Kornfeld, 112 East Chestnut St., Chicago 11, Executive Director.

AMERICAN DERMATOLOGICAL ASSOCIATION, Lake Placid Club, Lake Placid, N. Y., June 9-13. Dr. Louis A. Brunsting, 102 Second Ave. S.W., Rochester, Minn., Secretary.

AMERICAN DIABETES ASSOCIATION, Hotel Commodore, New York, May 30-31. Dr. John A. Reed, 11 West 42d St., New York 36, Secretary.

AMERICAN FEDERATION FOR CLINICAL RESEARCH, Haddon Hall, Atlantic City, N. J., May 3. Dr. Lawrence E. Hinkle Jr., 525 East 68th St., New York 21, Secretary.

AMERICAN GASTRO-ENTEROLOGICAL ASSOCIATION, Claridge Hotel, Atlantic City, N. J., May 1-2. Dr. H. Marvin Pollard, University Hospital, Ann Arbor, Mich., Secretary.

AMERICAN GERIATRICS SOCIETY, Hotel Commodore, New York, May 28-30. Dr. Malford W. Thewlis, 25 Mechanic St., Wakefield, R. I., Secretary.

AMERICAN GOITER ASSOCIATION, Drake Hotel, Chicago, May 7-9. Dr. George C. Shivers, 100 E. St. Vrain St., Colorado Springs, Colo., Secretary.

AMERICAN GYNECOLOGICAL SOCIETY, Lake Placid Club, Lake Placid, N. Y., June 15-17. Dr. John I. Brewer, 104 South Michigan Ave., Chicago, Secretary.

AMERICAN MEDICAL WOMEN'S ASSOCIATION, Barbizon-Plaza Hotel, New York, May 29-31. Dr. Elizabeth R. Fischer, 10848 South Fairfield Ave., Chicago 43, Secretary.

AMERICAN LARYNGOLOGICAL ASSOCIATION, Roosevelt Hotel, New Orleans, April 26-27. Dr. Harry P. Schenck, 326 South 19th St., Philadelphia 3, Secretary.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, Roosevelt Hotel, New Orleans, April 28-30. Dr. C. Stewart Nash, 277 Alexander St., Rochester, N. Y., Secretary.

AMERICAN NEUROLOGICAL ASSOCIATION, Hotel Claridge, Atlantic City, N. J., June 15-17. Dr. H. Houston Merritt, 710 West 168th St., New York 32, Secretary.

AMERICAN OPHTHALMOLOGICAL SOCIETY, The Homestead, Hot Springs, Va., May 28-30. Dr. Maynard C. Wheeler, 30 West 59th St., New York 19, Secretary.

AMERICAN ORTHOPEDIC ASSOCIATION, The Homestead, Hot Springs, Va., June 29-July 1. Dr. George O. Eaton, 4 East Madison St., Baltimore 2, Secretary.

AMERICAN OTOLOGICAL SOCIETY, Roosevelt Hotel, New Orleans, May 1-2. Dr. John R. Lindsay, 950 East 59th Street, Chicago 37, Secretary.

AMERICAN PEDIATRIC SOCIETY, Hotel Traymore, Atlantic City, N. J., May 6-8. Dr. Alms C. McGuinness, 237 Medical Laboratories, University of Pennsylvania, Philadelphia 4, Secretary.

AMERICAN PROCTOLOGIC SOCIETY, Hotel Statler, Boston, June 10-13. Dr. Stuart T. Ross, 131 Fulton Ave., Hempstead, N. Y., Secretary.

AMERICAN PSYCHIATRIC ASSOCIATION, Hotel Statler, Los Angeles, May 4-9. Dr. R. Finley Gayle Jr., 6300 Three Chopt Road, Richmond, Va., Secretary.

AMERICAN RHEUMATISM ASSOCIATION, Waldorf-Astoria, New York, May 28-29. Dr. William H. Kammerer, 33 East 61st St., New York 21, Secretary.

AMERICAN SOCIETY FOR CLINICAL INVESTIGATION, Chalfonte-Haddon Hall, Atlantic City, N. J., May 4. Dr. William M. M. Kirby, University of Washington School of Medicine, Seattle 5, Secretary.

AMERICAN SOCIETY OF MAXILLOFACIAL SURGEONS, Cleveland, April 26-29. Dr. Casper M. Epsteen, 25 East Washington St., Chicago 2, Secretary.

AMERICAN SOCIETY FOR THE STUDY OF STERILITY, Henry Hudson Hotel, New York, May 25-31. Dr. Walter W. Williams, 20 Magnolia Terrace, Springfield 8, Mass., Secretary.

AMERICAN THERAPEUTIC SOCIETY, The Biltmore, New York, May 28-31. Dr. Oscar B. Hunter Jr., 915 Nineteenth St. N.W., Washington 6, D. C., Secretary.

AMERICAN TRUDEAU SOCIETY, Hotel Statler, Los Angeles, May 18-22. Dr. John D. Steele, 1790 Broadway, New York 19, Secretary.

AMERICAN UROLOGICAL ASSOCIATION, Hotel Jefferson, St. Louis, May 11-14. Dr. Charles H. deT. Shivers, Boardwalk National Arcade Bldg., Atlantic City, N. J., Secretary.

ARIZONA MEDICAL ASSOCIATION, Pioneer Hotel, Tucson, April 26-30. Dr. D. W. Melick, 541 Security Bldg., Phoenix, Secretary.

ASSOCIATION OF AMERICAN PHYSICIANS, Haddon Hall, Atlantic City, N. J., May 5-6. Dr. W. Barry Wood Jr., 600 S. Kingshighway Blvd., St. Louis 10, Secretary.

CALIFORNIA MEDICAL ASSOCIATION, Biltmore Hotel, Los Angeles, May 24-28. Dr. Albert C. Daniels, 450 Sutter St., San Francisco 8, Secretary.

CATHOLIC HOSPITAL ASSOCIATION OF THE UNITED STATES AND CANADA, Kansas City, Mo., May 25-28. Rev. John J. Flanagan, S.J., 1438 South Grand Blvd., St. Louis 4, Director.

CONFERENCE OF PRESIDENTS AND OTHER OFFICERS OF STATE MEDICAL ASSOCIATIONS, New York, May 31. Mr. Thomas R. O'Brien, 634 North Grand Blvd., St. Louis 3, Secretary.

CONNECTICUT STATE MEDICAL SOCIETY, Hampden High School, New Haven, April 27-29. Dr. Creighton Barker, 160 St. Ronan St., New Haven, Secretary.

FLORIDA MEDICAL ASSOCIATION, Hollywood Beach Hotel, Hollywood, April 26-29. Dr. Samuel M. Day, 413 Professional Bldg., Jacksonville, Secretary.

GEORGIA, MEDICAL ASSOCIATION OF, Savannah, May 10-13. Dr. David H. Poer, 875 West Peachtree St. N.E., Atlanta, Secretary.

HAWAII TERRITORIAL MEDICAL ASSOCIATION, Island of Maui, April 30-May 3. Dr. Samuel L. Yee, 1163 South Beretania St., Honolulu, Secretary.

IDAHO STATE MEDICAL ASSOCIATION, Sun Valley, June 14-17. Dr. Robert S. McKean, 305 Sun Bldg., Boise, Secretary.

ILLINOIS STATE MEDICAL SOCIETY, Hotel Sherman, Chicago, May 19-22. Dr. Harold M. Camp, 224 South Main St., Monmouth, Secretary.

IOWA STATE MEDICAL SOCIETY, Des Moines, April 26-29. Dr. Allan B. Phillips, 529 36th St., Des Moines 12, Secretary.

KANSAS MEDICAL SOCIETY, Wichita, May 4-7. Dr. Dale D. Vermillion, 315 West Fourth St., Topeka, Secretary.

LOUISIANA STATE MEDICAL SOCIETY, Roosevelt Hotel, New Orleans, May 7-9. Dr. C. Grenes Cole, 1430 Tulane Ave., New Orleans 12, Secretary.

MARYLAND, MEDICAL AND CHIRURGICAL FACULTY OF THE STATE OF, Baltimore, April 28-29. Dr. George H. Yeager, 1211 Cathedral St., Baltimore 1, Secretary.

MASSACHUSETTS MEDICAL SOCIETY, Hotel Statler, Boston, May 19-21. Dr. Robert W. Buck, 22 The Fenway, Boston 15, Acting Secretary.

MEDICAL LIBRARY ASSOCIATION, New House Hotel, Salt Lake City, June 16-19. Miss Louise C. Lage, Lilly Research Laboratories, 740 South Alabama St., Indianapolis 6, Secretary.

MINNESOTA STATE MEDICAL ASSOCIATION, Hotel St. Paul, St. Paul, May 18-20. Dr. B. B. Souster, 496 Lowry Medical Arts Bldg., St. Paul 2, Secretary.

MISSISSIPPI STATE MEDICAL ASSOCIATION, Biloxi, May 11-14. Mr. Roland B. Kennedy, 508 First Federal Bldg., Jackson, Executive Secretary.

MISSOURI STATE MEDICAL ASSOCIATION, Kansas City, April 26-29. Dr. H. E. Petersen, 634 N. Grand Blvd., St. Louis 3, Secretary.

NATIONAL TUBERCULOSIS ASSOCIATION, Statler and Biltmore hotels, Los Angeles, May 18-22. Mr. Kemp D. Battle, 1790 Broadway, New York 19, Secretary.

NEBRASKA STATE MEDICAL ASSOCIATION, Paxton Hotel, Omaha, May 11-14. Dr. R. B. Adams, 1315 Sharp Bldg., Lincoln 8, Secretary.

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NEW JERSEY, MEDICAL SOCIETY OF, Haddon Hall, Atlantic City, May 17-20. Dr. Marcus H. Greiffinger, 315 West State St., Trenton 8, Secretary.

NEW MEXICO MEDICAL SOCIETY, Hilton Hotel, Albuquerque, May 7-9. Dr. T. E. Kircher Jr., 221 West Central Ave., Albuquerque, Secretary.

NEW YORK, MEDICAL SOCIETY OF THE STATE OF, Hotel Statler, Buffalo, May 4-8. Dr. Walter P. Anderton, 386 Fourth Avenue, New York 16, Secretary.

NORTH CAROLINA, MEDICAL SOCIETY OF THE STATE OF, Hotel Carolina, Pinehurst, May 10-13. Dr. Millard D. Hill, 203 Capital Club Bldg., Raleigh, Secretary.

NORTH DAKOTA STATE MEDICAL ASSOCIATION, Clarence Parker Hotel, Minot, May 9-12. Dr. E. H. Boerth, Box 1198, Bismarck, Secretary.

OGDEN SURGICAL SOCIETY, Orpheum Theater, Ogden, Utah, May 20-22. Dr Irwin B. McQuarrie, 401 Eccles Bldg., Ogden, Utah, President.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY, Los Angeles, May 24-28, Dr. H. P. House, 1136 West 8th St., Los Angeles 14, Secretary.

RHODE ISLAND MEDICAL SOCIETY, Providence, May 6-7. Dr. Thomas Perry Jr., 106 Francis Street, Providence, Secretary.

SOCIETY OF BIOLOGICAL PSYCHIATRY, Hotel Statler, Los Angeles, May 3. Dr. George N. Thompson, 2010 Wilshire Blvd., Los Angeles 5, Secretary.

SOCIETY OF CLINICAL SURGERY, Spring Meeting, Nashville, Tenn., May 1-2. Dr. Champ Lyons. 620 South 20th St., Birmingham 5, Ala., Secretary.

SOCIETY FOR INVESTIGATIVE DERMATOLOGY, Belmont Plaza Hotel, New York, May 30-31. Dr. Herman Beerman, 255 South 17th St., Philadelphia 3, Secretary.

SOCIETY FOR PEDIATRIC RESEARCH, Hotel Traymore, Atlantic City, N. J., May 4-6. Dr. Sydney S. Gellis, 330 Brookline Ave., Boston 15, Secretary.

SOCIETY FOR VASCULAR SURGERY, Hotel Pierre, New York, May 31. Dr. George D. Lilly, 25 S.E. Second Ave., Miami 32, Fla., Secretary.

SOUTH CAROLINA MEDICAL ASSOCIATION, Columbia Hotel, Columbia, May 4-7. Dr. Robert Wilson Jr., 120 W. Cheves St., Florence, Secretary.

SOUTH DAKOTA STATE MEDICAL ASSOCIATION, Alex Johnson Hotel, Rapid City, June 14-16. Dr. G. I. W. Cottam, 300 First National Bank Bldg., Sioux Falls, Secretary.

SOUTHEASTERN ALLERGY ASSOCIATION, Andrew Jackson Hotel, Nashville, Tenn., May 15-16. Dr. Katharine B. MacInnis, 1515 Bull St., Columbia 1, S. C., Secretary.

SOUTHERN OREGON MEDICAL SOCIETY, Oregon Caves Chateau, Oregon Caves, Ore., June 10. Dr. R. Ray Johnson, Grants Pass, Ore., Secretary.

SOUTHWEST ALLERGY FORUM, Hotel Muehlebach, Kansas City, Mo., June 14-16. Dr. Frederic Speer, 2601 Parallel Ave., Kansas City 4, Kans., Secretary.

STUDENT AMERICAN MEDICAL ASSOCIATION, Edgewater Beach Hotel, Chicago, June 15-17. Mr. Russell F. Staudacher, 535 North Dearborn St., Chicago 10, Executive Secretary.

TEXAS MEDICAL ASSOCIATION, Shamrock Hotel, Houston, April 26-29. Mr. N. C. Forrester, 1801 Lamar Blvd., Austin, Executive Secretary.

THE ENDOCRINE SOCIETY, Hotel Statler, New York, May 28-30. Dr. Henry H. Turner, 1200 North Walker St., Oklahoma City 3, Secretary.

U. S. CHAPTER, INTERNATIONAL COLLEGE OF SURGEONS, SURGICAL DIVISION MEETINGS:

CHICAGO, Congress Hotel, May 5-6. Dr. Karl A. Meyer, 30 North Michigan Ave., Chicago 2, Chairman.

UPPER PENINSULA MEDICAL SOCIETY, Escanaba, Mich., June 19-20. Dr. N. L. Lindquist, 1103 Ludington St., Escanaba, Mich., Secretary.

WESTERN SECTION AMERICAN UROLOGICAL ASSOCIATION, Palace Hotel, San Francisco, April 27-30. Dr. A. A. Kutzmann, 1930 Wilshire Blvd., Los Angeles 5, Secretary.

WESTERN SOCIETY OF ELECTRO-ENCEPHALOGRAPHY, Hotel Statler, Los Angeles, May 9-10. Dr. Sylvester N. Berens, 902 Boren Ave., Seattle, Secretary.

WYOMING STATE MEDICAL SOCIETY, Henning Hotel, Casper, June 11-13. Dr. Glenn W. Koford, P. O. Box 1252, Cheyenne, Secretary.

FOREIGN

BRITISH MEDICAL ASSOCIATION, Cardiff, S. Wales, July 13-17. Dr. A. MacRae, B. M. A. House, Tavistock Square, London W.C.1, England, Secretary.

CANADIAN MEDICAL ASSOCIATION, Winnipeg, Manitoba, Canada, June 15-19. Dr. T. C. Routley, 135 St. Clair Avenue W., Toronto 5, Ontario, Canada, General Secretary.

CONGRESS OF INTERNATIONAL ANESTHESIA RESEARCH SOCIETY, Chateau Frontenac, Quebec, Canada, October 26-29. Dr. A. William Friend, 515 Nome Ave., Akron 20, Ohio, Chairman, Program Committee.

CONGRESS OF INTERNATIONAL LEAGUE AGAINST RHEUMATISM, Geneva and Zurich, Switzerland, Aug. 24-29. For information write: Dr. W. Tegner, The London Hospital, London E.1, England.

CONGRESS OF THE INTERNATIONAL SOCIETY OF ANGIOLOGY, Lisbon, Portugal, Sept. 18-20. Dr. Henry Haimovici, 105 East 90th St., New York 28, N. Y., U. S. A., Secretary.

CONGRESS OF THE INTERNATIONAL SOCIETY OF SURGERY, Lisbon, Portugal, Sept. 14-20. Dr. L. Dejardin, 141, rue Belliard, Brussels, Belgium, General Secretary.

EUROPEAN CONGRESS OF ALLERGOLOGY, Copenhagen, Denmark, May 20-23. Dr. Egon Brunn, Gersonavej 8, Hellerup, Copenhagen, Denmark, Secretary General.

INTERNATIONAL CONFERENCE ON THROMBOSIS AND EMBOLISM, Basle, Switzerland, July 15-19, 1954. Dr. W. Merz, Chief Medical Officer, Gynecological Clinic, University of Basle, Basle, Switzerland, Hon. Secretary.

INTERNATIONAL CONGRESS OF AUDIOLOGY, Leiden, Netherlands, June 5-6. Dr. H. A. E. ve Dishoeck, Leiden University, Leiden, Netherlands, President.

INTERNATIONAL CONGRESS OF ELECTROENCEPHALOGRAPHY AND CLINICAL NEUROPHYSIOLOGY, Boston, Mass., U. S. A., Aug. 18-21. Dr. Robert S. Schwab, Massachusetts General Hospital, Boston 14, Mass., U. S. A., Secretary-General.

INTERNATIONAL CONGRESS OF THE EUROPEAN SOCIETY OF HAEMATOLOGY, Amsterdam, Holland, Sept. 8-12. Dr. M. C. Verloop, Mallesingle 15, Utrecht, Holland, Secretary.

INTERNATIONAL CONGRESS ON GENETICS, Bellagio, Italy, August 4. Prof. C. Barigozzi, Instituto de Genetica, Universita de Milano, 10 via Celoria, Milan, Italy, Secretary.

INTERNATIONAL CONGRESS OF GYNECOLOGY, Geneva, Switzerland, July 21-26, 1954. Dr. Maurice Fabre, 1, rue Jules-Lefebore, Paris IXe, France, General Secretary.

INTERNATIONAL CONGRESS OF HIPPOCRATIC MEDICINE, Evian, France, Sept. 3-6. Prof. P. Delore, 13 rue Jarente, Lyons, France, Secretary-General.

INTERNATIONAL CONGRESS FOR HISTORY OF SCIENCE, Jerusalem, Israel, August 3-7. Prof. F. S. Bodenheimer, Hebrew University, Jerusalem, Israel, President.

INTERNATIONAL CONGRESS OF INTERNATIONAL COLLEGE OF SURGEONS, Sao Paulo, Brazil, April 26-May 2, 1954. Dr. Max Thorek, 1516 Lake Shore Drive, Chicago, Illinois, U.S.A., Secretary-General.

INTERNATIONAL CONGRESS OF LOGOPEDICS AND PHONIATRICS, Milan and Stresa, Italy, Sept 1-7. Dr. Deso A. Weiss, 115 East 86th St., New York 28, N. Y., U. S. A., General Secretary.

INTERNATIONAL CONGRESS ON MEDICAL LIBRARIANSHIP, London, England, July 20-25. Mr. W. R. LeFanu, % London School of Hygiene and Tropical Medicine, Keppel Street, London, W.C.1, England, Chairman.

INTERNATIONAL CONGRESS ON MENTAL HEALTH, University of Toronto, Toronto, Ontario, Canada, Aug. 14-21, 1954. For information write: Executive Officer, International Congress on Mental Health, 111 St. George St., Toronto, Ontario, Canada.

INTERNATIONAL CONGRESS OF MICROBIOLOGY, Rome, Italy, Sept 6-12. For information write: Dr. V. Puntoni, Citta Universitaria, Rome, Italy.

INTERNATIONAL CONGRESS ON OBSTETRICS AND GYNECOLOGY, Geneva, Switzerland, July 20-24, 1954. For information write: Prof. H. de Watteville, Maternité Hôpital Cantonal, Geneva, Switzerland.

INTERNATIONAL CONGRESS OF OTO-NEURO-OPHTHALMOLOGY, Bologna, Italy, May 3-7. Dr. Guiseppe Cristini, Clinica Oculistica, Policlinico, Bologna, Italy, General Secretary.

INTERNATIONAL CONGRESS OF OTORHINOLARYNGOLOGY, Amsterdam, Netherlands, June 8-13. Dr. W. H. Struben, J. J. Viottastraat 1, Amsterdam, Netherlands, Secretary.

INTERNATIONAL CONGRESS OF PAEDIATRICS, Havana, Cuba, Oct. 12-17. Prof. Felix Hurtado, 5a Avenue 124, Miramar, Havana, Cuba President.

INTERNATIONAL CONGRESS OF RADIOLOGY, Copenhagen, Denmark, July 19-25. Professor Flemming Norgaard, 10 Oster Voldgade, Copenhagen K, Denmark, Secretary General.

INTERNATIONAL CONGRESSES OF TROPICAL MEDICINE AND MALARIA, Istanbul, Turkey, Aug. 28-Sept. 4. Professor Dr. Ihsan Sükrü Aksel, Tunel Meydam, Beyoglu, Istanbul, Turkey, General Secretary.

INTERNATIONAL CONVENTION OF X-RAY TECHNICIANS, Royal York Hotel, Toronto, Canada, June 28-July 2. For information write: Miss Beatrice Hurley, R.T., Registrar, St. Catherine Hospital, East Chicago, Ind., U. S. A

INTERNATIONAL FERTILITY ASSOCIATION, Henry Hudson Hotel, New York, N. Y., U. S. A., May 25-31. Dr. Abner I. Weisman, 1160 Fifth Avenue, New York 29, N. Y. U. S. A., Associate Secretary General.

INTERNATIONAL GERONTOLOGICAL CONGRESS, London and Oxford, England, July 12-22, 1954. Prof. R. E. Tunbridge, General Infirmary, Department of Medicine, The University, Leeds, England, President.

INTERNATIONAL GYNAECOLOGICAL MEETING, Paris, France, May 22-23. For information write: Dr. Jacques Courtois, 1, rue Racine, St. Germain-en-Laye, Seine et Oise, France.

INTERNATIONAL HOSPITAL CONGRESS, London, England, May 25-30. Capt. J. E. Stone, 10 Old Jewry, London, EC2, England, Hon. Secretary.

INTERNATIONAL LEPROSY CONGRESS, Madrid, Spain, Oct. 3-10. Dr. Felix Contreras, Moreto 15, Madrid, Spain, Secretary.

INTERNATIONAL PHYSIOLOGICAL CONGRESS, Montreal, Canada, Aug. 31-Sept. 4. Dr. A. S. V. Burgen, Dept. of Physiology, McGill University, Montreal, Canada, Secretary.

INTERNATIONAL PSYCHO-ANALYTICAL CONGRESS, Bedford College, Regent's Park, London N.W.1, England, July 26-30. Dr. Ruth S. Eissler, 285 Central Park West, New York 24, N. Y., Hon. Secretary.

INTERNATIONAL VETERINARY CONGRESS, Stockholm, Sweden, Aug. 9-15. Prof. Axel Isaksson, Institute of Veterinary Medicine, Stockholm 50, Sweden, Secretary.

PACIFIC SCIENCE CONGRESS, Quezon City and Manila, Philippines, Nov. 16-28. Dr. Patrocinio Valenzuela, College of Pharmacy, University of the Philippines, Quezon City, Philippines, Secretary-General.

PAN AMERICAN CONGRESS OF THE MEDICAL PRESS, Buenos Aires, Argentine, July 12-16. Secretaria del Congress, 763 Uriburu, Buenos Aires, Argentine.

ROYAL SANITARY INSTITUTE HEALTH CONGRESS, Hastings, England, April 28-May 1. For information write: Secretary, Royal Sanitary Institute, 90 Buckingham Palace Road, London 5, W1, England.

WORLD CONFERENCE ON MEDICAL EDUCATION, British Medical Association House, Tavistock Square, W.C.1, London, England, Aug. 22-29. Secretariat: World Medical Association, 2 East 103d St., New York 29, N. Y., U. S. A.

WORLD CONGRESS OF THE WORLD CONFEDERATION FOR PHYSICAL THERAPY, London, England, Sept. 7-12. Miss M. J. Neilson, Chartered Society of Physiotherapy, Tavistock House, South, Tavistock Square, London, W.C.1, England, Secretary.

WORLD MEDICAL ASSOCIATION, The Hague, Netherlands, Aug. 31-Sept. 7. Dr. Louis H. Bauer, 2 East 103d St., New York 29, N. Y., Secretary-General.

DEATHS

Moloy, Howard Carman ● New York City; born in Thedford, Ontario, Canada, in 1903; University of Western Ontario Faculty of Medicine, London, Ontario, Canada, 1927; assistant clinical professor of obstetrics and gynecology at Columbia University College of Physicians and Surgeons; formerly on the faculty of his alma mater; fellow of the American Gynecological Society and the New York Obstetrical Society; specialist certified by the American Board of Obstetrics and Gynecology; with the late Dr. William E. Caldwell and Dr. D. Anthony D'Esopo wrote the section on forceps operation in "Nelson's New Loose-Leaf Surgery: Gynecology; Surgical Obstetrics"; contributed to the section on pelvis morphology and mechanism of labor in Dr. Carl H. Davis' "System of Gynecology and Obstetrics"; author of "Clinical and Roentgenologic Evaluation of the Pelvis in Obstetrics" published in 1951 by W. B. Saunders; contributed to many of the standard textbooks in those sections relating to pelvic shape and influence on labor; together with the late Dr. William E. Caldwell, developed the precision stereoscope, a machine that shows graphically, by means of stereoscopically superimposed x-rays, the relation of size, shape, and position of the fetal head and the maternal pelvis at the start of labor; assistant attending obstetrician and gynecologist and assistant clinical professor of obstetrics and gynecology at the Columbia-Presbyterian Medical Center, where he died March 13, aged 50, of a heart attack.

Baruch, Herman Benjamin, Wyandanch, N. Y.; born in Camden, S. C., April 28, 1872; College of Physicians and Surgeons, medical department of Columbia College, New York, 1895; practiced medicine in New York from 1895 to 1903, when he became a member of the stock exchange firm of Baruch Brothers; in 1918 the concern was merged with M. Hentz & Co., bankers and commission merchants, of which he became senior partner; served as special representative of the board of economic warfare in Brazil and as special economic adviser to the U. S. ambassador to Brazil in 1943; ambassador extraordinary and plenipotentiary of the United States to Portugal from 1945 to 1947, when he became ambassador extraordinary and plenipotentiary to the Netherlands, serving until 1949; in 1949 awarded Grand Cross of the Royal Order of the Netherlands Lion and in 1950 Grand Cross of the Order of Christus (Portuguese); in 1932 a democratic presidential elector; member of the New York Academy of Medicine, American Arbitration Association, and Alien Enemy Review Board; member of the alumni associations of the University of Virginia, City College of New York, and Sloane Maternity and Mount Sinai hospitals; director of Phi Beta Kappa and Kappa Sigma; president of the Doctor Simon Baruch Foundation; owner of the Bagetelle Nursery in Huntington, L. I.; died March 15, aged 80, of coronary thrombosis.

Wilkins, Walter E. ● Jacksonville, Fla.; born in Columbia, S. C., July 9, 1903; Vanderbilt University School of Medicine, Nashville, Tenn., 1938; fellow of the American Public Health Association; member of Phi Beta Pi and Alpha Omega Alpha; since 1940 on the governing council of the American School Health Association and its president in 1946-1947; at one time on the faculty of his alma mater; director of the division of school health, North Carolina State Board of Health, Raleigh, from 1939 to 1942; medical nutrition officer, War Food Administration, Washington, D. C., from 1942 to 1945; senior surgeon in the U. S. Public Health Service Reserve, in charge of nutrition section in 1945; director of nutrition investigations and services, Florida State Board of Health, from 1946 to 1950; inventor of the cup needle for taking blood samples for laboratory tests; died March 2, aged 49.

Bolin, Zera Exley ● Captain, U. S. Army, retired, San Francisco; Jefferson Medical College of Philadelphia, 1914; also a graduate in pharmacy; served during World War I; entered the medical corps of the U. S. Army in March, 1918; retired in December, 1922, for disability in line of duty; specialist certified by the American Board of Pathology; member of the American Association of Pathologists and Bacteriologists, Association of Military Surgeons of the United States, College of American Pathologists, American Society of Clinical Pathologists, National Gastroenterological Association, and the American Public Health Association; at one time assistant professor of pathology at the University of California Medical School; served on the staffs of Mary's Help, St. Luke's, and the University of California hospitals; died Feb. 11, aged 64, of arteriosclerotic heart disease.

Macnie, John Silliman ● Minneapolis; born in Newburg, N. Y., May 21, 1874; Columbia University College of Physicians and Surgeons, New York, 1896; formerly associate professor of ophthalmology at the University of Minnesota Medical School and associate professor of ophthalmology and otology at the University of Minnesota Graduate School; specialist certified by the American Board of Ophthalmology and the American Board of Otolaryngology; member of the American Academy of Ophthalmology and Otolaryngology; fellow of the American College of Surgeons; on the staffs of the Northwestern and St. Barnabas hospitals; died Jan. 30, aged 78, of cerebral thrombosis, arteriosclerosis, and acute bronchopneumonia.

Scheer, Max, New York City; born in 1883; Columbia University College of Physicians and Surgeons, New York, 1905; clinical professor of dermatology and syphilology at the Post-Graduate Medical School of the New York University-Bellevue Medical Center; specialist certified by the American Board of Dermatology and Syphilology; member of the American Academy of Dermatology and Syphilology; fellow of the New York Academy of Medicine; served on the staffs of Mount Sinai, University, Bellevue, and Harlem hospitals; died in Syracuse March 1, aged 69.

Brenneman, Richard Emmor ● Glendale, Calif.; Harvard Medical School, Boston, 1900; an Associate Fellow of the American Medical Association; specialist certified by the American Board of Radiology; member of the American Society of Anesthesiologists and the American College of Radiology; at one time practiced in Pittsburgh, where he was on the staffs of the Passavant and Presbyterian hospitals; on the courtesy staff of the Glendale Sanitarium and Hospital; died in the VA Hospital, Los Angeles, Feb. 15, aged 79, of encephalomalacia.

Abel, Rudolph N., Los Angeles; St. Louis College of Physicians and Surgeons, 1906; died Jan. 19, aged 77, of intestinal cancer and arteriosclerotic heart disease.

Allhands, Frank Dallas, Crawfordsville, Ind.; Kentucky School of Medicine, Louisville, 1897; served on the staff of Montgomery County Culver Union Hospital; died Jan. 7, aged 83, of cerebral hemorrhage and arteriosclerosis.

Amesbury, Ivon Cuthbert Raleigh ● Boston; Boston University School of Medicine, 1890; died Jan. 12, aged 83, of arteriosclerotic heart disease.

Blair, George Dewey ● Pittsburgh; Jefferson Medical College of Philadelphia, 1923; died in St. Francis Hospital Dec. 31, aged 54, of hypertension and carcinoma of the bladder.

Blumenthal, Herman Joseph, Brooklyn; Yale University School of Medicine, New Haven, Conn., 1921; died Nov. 30, aged 56, of a heart attack.

Connelley, Walter Constantine, Salyersville, Ky.; University of Louisville Medical Department, 1907; died Dec. 18, aged 73, of cancer.

Crane, James Wilder ● Baring, Maine; Medical School of Maine, Portland, 1908; died Dec. 5, aged 73, of chronic myocarditis and coronary thrombosis.

● Indicates Member of the American Medical Association.

Criso, Bruce Lamont, Wilmington, Calif.; University of Southern California College of Medicine, Los Angeles, 1903; died Jan. 25, aged 73, of coronary thrombosis.

Current, Howard Wesley ✪ Montoursville, Pa.; Jefferson Medical College of Philadelphia, 1925; a veteran of World War I; served as president of the board of health; affiliated with Williamsport (Pa.) Hospital; died Jan. 2, aged 56, of myelogenous leukemia.

Davis, Francis Marion, Canton, N. C.; University of Nashville (Tenn.) Medical Department, 1894; Vanderbilt University School of Medicine, Nashville, 1894; died Jan. 15, aged 82, of coronary disease.

Davis, John Gibson ✪ Roanoke, Va.; Medical College of Virginia, Richmond, 1885; died Jan. 31, aged 89, of influenza and cardiac failure.

Davison, Guy W., Greencastle, Pa.; Medico-Chirurgical College of Philadelphia, 1903; died Jan. 7, aged 79.

DeWitt, Charles Herman Jr. ✪ Macedonia, Iowa; John A. Creighton Medical College, Omaha, 1910; died in Oakland Jan. 15, aged 66, of myocardial degeneration and asthma.

First, Francis Ray ✪ Checotah, Okla.; University Medical College of Kansas City, Mo., 1911; veteran of World War I; served on the staffs of the Oklahoma Baptist and Muskogee General hospitals in Muskogee; died in St. John's Hospital, Tulsa, Jan. 31, aged 65, of uremia.

Gifford, William Royal ✪ East Orange, N. J.; University and Bellevue Hospital Medical College, New York, 1918; died Jan. 10, aged 60, of coronary embolism and cerebral embolism.

Gilbert, John Dunham, Carlton, Minn.; College of Physicians and Surgeons of Chicago, 1890; died in Raiter Hospital, Cloquet, Jan. 17, aged 88, of bronchopneumonia.

Gray, Stoddard Percy ✪ Chester, Pa.; Jefferson Medical College of Philadelphia, 1904; served during World War I; died Dec. 13, aged 75, of leukemia.

Gumbiner, Alfred Albert, Los Angeles; Long Island College Hospital, Brooklyn, 1902; died in the Good Samaritan Hospital Dec. 3, aged 72, of pulmonary embolism and myocardial infarction.

Harman, William Roby, Springfield, Mo.; American Medical College, St. Louis, 1903; died Jan. 27, aged 86.

Harris, Meyer Mathews, New York City; Cornell University Medical College, New York, 1920; member of the Endocrine Society and the American Psychosomatic Society; principal research internist, New York State Psychiatric Institute; died Dec. 22, aged 59, of coronary thrombosis.

Herzberg, Mortimer, Wilmington, N. C.; University of Pennsylvania Department of Medicine, Philadelphia, 1902; specialist certified by the American Board of Pathology; member of the College of American Pathologists; at one time on the staff of the Jewish Hospital in Philadelphia; died in James Walker Memorial Hospital Jan. 7, aged 73, of coronary thrombosis.

Holst, Claude Frederick, Little Falls, Minn.; University of Minnesota Medical School, Minneapolis, 1901; died in St. Gabriel's Hospital Jan. 12, aged 78, of hypostatic pneumonia, bronchiectasis, and myocardosis.

Kelly, Jon Nelson ✪ La Porte, Ind.; Central College of Physicians and Surgeons, Indianapolis, 1898; veteran of the Spanish-American War; formerly city and county health officer; past president of the Northern Tri-State Medical Association; affiliated with Fairview Hospital and the Holy Family Hospital, where he died Jan. 20, aged 76, of nephritis, myocarditis, and arteriosclerosis.

Kosik, Constantine Austin ✪ New York City; University of Buffalo School of Medicine, 1922; died Jan. 27, aged 55, of a heart attack.

Lazenby, Earl Kilpatrick ✪ Fayetteville, N. C.; Atlanta Medical College, 1914; formerly affiliated with the Veterans Administration; died Jan. 17, aged 66, of portal cirrhosis and diabetes mellitus.

Liljencrantz, Guy Homer, Watsonville, Calif.; Rush Medical College, Chicago, 1901; past president of the Alameda County Medical Society; affiliated with Peralta Hospital in Oakland; died Jan. 27, aged 76, of arteriosclerotic heart disease.

Luginbuhl, Christian Bateman ✪ Des Moines, Iowa; Rush Medical College, Chicago, 1914; specialist certified by the American Board of Internal Medicine; fellow of the American College of Physicians; past president of the Polk County Medical Society; member of the Iowa Commission on Industrial Health; medical advisor to Iowa industrial commissioner; affiliated with Iowa Lutheran Hospital, Broadlawns General Hospital, and the Iowa Methodist Hospital, where he died Jan. 16, aged 70, of adenocarcinoma.

McCartney, William Henry ✪ Des Moines, Iowa; Chicago Homeopathic Medical College, 1904; died Jan. 1, aged 77, of coronary embolism.

McDonald, James Edward ✪ Mason City, Iowa; Keokuk Medical College, 1893; also a pharmacist; served during World War I; for many years county coroner and city health officer; died Jan. 23, aged 85, of uremia.

Mace, Lloyd Russell, Los Angeles; Jefferson Medical College of Philadelphia, 1905; for many years physician for the California State Athletic Commission; died Jan. 23, aged 69, of cerebral hemorrhage.

McGee, R. Proctor ✪ Los Angeles; Denver College of Physicians and Surgeons, 1903; also a dentist; specialist certified by the American Board of Plastic Surgery and a member of the founders group; served overseas during World War I; affiliated with Presbyterian Hospital-Olmsted Memorial; died Jan. 14, aged 75, of cerebral thrombosis.

Marder, Sumner Norman, Bethesda, Md.; Yale University School of Medicine, New Haven, Conn., 1947; certified by the National Board of Medical Examiners; specialist certified by the American Board of Radiology; senior assistant surgeon in the U. S. Public Health Service Reserve, engaged in cancer research at the National Institute of Health; died March 4, aged 30.

Mogan, Christopher Joseph ✪ Memphis, Tenn.; Jefferson Medical College of Philadelphia, 1929; member of the Medical Society of the State of Pennsylvania; served during World War II; affiliated with the Veterans Administration; died Dec. 23, aged 50, of lymphosarcoma.

Moore, Daniel Milton ✪ Monroe, La.; Atlanta College of Physicians and Surgeons, 1913; specialist certified by the American Board of Radiology; member of the Radiological Society of North America and the American College of Radiology; affiliated with E. A. Conway Memorial Hospital and St. Francis Sanitarium, where he died Jan. 7, aged 69, of cerebral hemorrhage.

Morse, Frank Le Forest ✪ St. Louis; Beaumont Hospital Medical College, St. Louis, 1900; affiliated with Missouri Baptist, Christian, and Evangelical Deaconess hospitals, and the Park Lane Memorial Hospital, where he died Jan. 14, aged 76.

Morton, George D. ✪ Honey Brook, Pa.; University of Pennsylvania Department of Medicine, Philadelphia, 1892; at one time on the faculty of his alma mater; served on the school board; affiliated with Coatesville (Pa.) Hospital; member of the board of the Chester County Health and Tuberculosis Society, of which he was vice-president for many years; surgeon for the Pennsylvania Railroad; died Jan. 29, aged 81, of injuries received when struck by an automobile.

Norrick, John Howard, Oklahoma City, Okla.; Starling Medical College, Columbus, 1892; died Jan. 14, aged 85, of influenza.

Slater, Maxwell Ehron ✪ New York City; Long Island College Hospital, Brooklyn, 1905; specialist certified by the American Board of Ophthalmology; on the staff of the Good Samaritan Hospital; died Dec. 30, aged 70.

Wood, Orlin Pearl, Marysville, Kan.; University Medical College of Kansas City, Mo., 1900; county health officer; died Jan. 16, aged 73, of a heart attack.

FOREIGN LETTERS

JAPAN

Institute for Infectious Disease.—The Institute for Infectious Diseases, the University of Tokyo, celebrated its 60th anniversary Nov. 28-30, 1952. Hundreds of distinguished guests included Dr. Tadao Yanaibara, president of the University of Tokyo, professors, scientists, and officials representing various fields of science and medicine.

On the wall of the auditorium were hung the portraits of the directors since its foundation. The first director was the late Dr. Shibasaburo Kitasato, whose portrait was especially decorated with white ribbon, to denote that the occasion was also in remembrance of the 100th birthday of this great scientist. Among the persons attending was Dr. Zenjirô Kitasato, successor to the late professor and present director of the Kitasato Institute; another was His Excellency the Deputy-Ambassador from Germany.

The institute was established in 1892 under the auspices of Dr. Shinpei Goto, director of the sanitary board of the home department at that time, aided by Yukichi Fukuzawa, Ichizaemon Morimura, and Drs. Sensai Nagayo and Tai Hasegawa. The date of the foundation, therefore, was one year after the establishment of the Robert Koch Institute in Berlin and three years later than the Pasteur Institute in Paris. The special mission imposed on the Institute for Infectious Diseases has been perfectly carried out regardless of its administrative situation, whether it was under the jurisdiction of the Ministry for Home Affairs, Ministry of Education, or the University of Tokyo.

Examples of discoveries of pathogenic agents, taken from the speech by Dr. S. Hasegawa, present director of the institute are the discovery of plague bacillus by Kitasato in 1894, the dysentery bacillus by Kiyoshi Shiga in 1897, the varied forms of the dysentery bacillus by Kenzô Futaki in 1903, Spirocheaeta morsus-muris, the causative agent of ratbite fever, by Futaki and his associates in 1915, the pathogenic agent of tsutsugamushi fever by Mataro Nagayo and others in 1930, and the cause of lymphogranulomatosis by Yoneji Miyagawa. Other contributions to medical science and public health have been made by the members and associates of the institute especially in the fields of physiological and chemical bacteriology and in chemotherapeutics. The papers were published chiefly in the *Japanese Journal of Experimental Medicine*, the institute's official journal.

During the memorial meetings symposiums were held by the staff. The scientific exhibits showing recent achievements in the study of infectious diseases were open to the general public for three days. The institute now has 10 research divisions and 360 laboratory workers directed by research professors, assistant professors, and specialists. Each laboratory division has its own field of research: The laboratory of Prof. S. Hosoya is working on antibiotics and exotoxins and endotoxins. The laboratory of Prof. S. Hasegawa is engaged in research for antibacterial substances from various plants. Prof. N. Takeda's laboratory is interested in chemical research of bacterial constituents, chemotherapy of tuberculosis and leprosy, and tuberculin production. Prof. T. Nagano's laboratory is primarily in charge of virus research and betterment of rabies vaccine.

The laboratory of Prof. K. Ando is working on production of effective vaccines for immunization against enterobacteria and rickettsial infections. Pure serologic research is also being done. The division of Prof. S. Kudo is engaged in preservation of micro-organisms in collaboration with the International Federation of Culture Collection of Micro-organisms. Professor Miyazaki, pathology division, is working on experimental pathology, the chief interest being centered around inflammation and tissue culture in relation to nucleic acids. Assistant Prof. M. Sasa is studying medical entomology, especially with regard to classification and cultivation of the tick vector of tsutsugamushi fever. Prof. S. Akiya, biochemistry division, is investigating bacterial polysaccharides and antibacterial action of chemical compounds. Assistant Prof. S. Arakawa is in charge of study of the trachoma virus and purified smallpox vaccine and measles virus. Prof. O. Kitamoto, director of the hospital, is also in charge of the clinical laboratory. The effectiveness of various kinds of rabies vaccine is being investigated comparatively. Paralysis after rabies vaccination is being intensively studied.

Workers in bacteriology in the period of transition have devoted their efforts chiefly to culturing micro-organisms and animal experimentation, but the scope of bacteriology has now expanded to include every modern science. The institute is no exception in this expansion, and every effort is being made to conform with this progress and to meet the demands of public health in Japan.

PARIS

Allergic Dermatoses.—A. Tzank and E. Sidi discussed statistical studies on allergic dermatosis at the St. Louis Hospital Laboratory. Tests were made in 818, 799, and 761 cases in 1949, 1950, and 1951 respectively. Research techniques were perfected by the use of reagents not previously known to be sensitizing. The reagents were discovered by means of mucosal tests and by investigations at the patients' places of employment. The number of accidents due to sulfonamides and anesthetics have diminished in number, but those due to penicillin, streptomycin, and antihistaminic ointments have increased considerably. When hypersensitization occurs, its mechanism is as follows: When the patient applies "phenergan" (10-[2-dimethylamino-1-propyl] phenothiazine) cream locally, he becomes sensitive to "phenergan" administered orally. The sensitization reaction is intensified in summer by photosensitization on eczematous areas, especially areas not covered by clothing. When a tablet of "phenergan" is taken, the patient experiences a general as well as a cutaneous reaction; the temperature rises to 39 C (102.2 F) even with minute doses of "phenergan." On the other hand, persons sensitized by a given ointment do not necessarily become sensitive to other ointments containing antihistaminics. The authors reported the first case of acute dermatitis provoked by the local application of *p*-aminosalicylic acid. This sensitization to *p*-aminosalicylic acid involves sensitization to other "para" compounds. Patients thus sensitized cannot be treated with sulfonamide drugs nor be anesthesized with procaine hydrochloride (novocaine*). They must avoid physical contact with "para" dyes and objects dyed with an agent with one "para" group in the formula. Here also, sensitization by physical contact induces sensitization to the compound administered internally, and a very small dose of *p*-aminosalicylic acid can provoke very serious reactions. The authors insisted on the necessity of avoiding application on the skin of sensitizing products that later may be ingested internally, especially if they belong to a group of compounds having a primary amine function.

Sensitivity to Water and to Taenia Saginata.—Professor Valéry-Radot, P. Blamoutier, and Mrs. Holzer presented one case of urticaria and hemoclastic crisis subsequent to ingestion of water. The patient, a 57-year-old house-painter, had suffered from urticarial eruptions since 1948. It was found that the drinking of a glass of tap water or of 150 cc. of Vichy water provoked the appearance of urticaria on the arms, trunk, and legs. He experienced a feeling of heat and swelling of the face, and at the same time the leukocyte level decreased, once from 3,800 to 1,800 and another time from 7,800 to 5,000. Ingestion of water distilled twice produced no reaction. These authors believe that such persons are sensitized by ions present in water. P. Blamoutier presented the exceptional case of a 60-year-old

The items in these letters are contributed by regular correspondents in the various foreign countries.

woman who acquired a Taenia saginata cestode in Syria in 1929. After an emotional shock in 1950, this woman suffered from an asthma crisis, which yielded to antihelminthic treatment. Some months later, urticaria associated with eczema occurred, and at the same time Taenia saginata reappeared in the stools. After treatment by duodenal intubation and definite disappearance of the cestode, all morbid syndromes of the skin disappeared.

Infantile Eczema.—P. Voringer described the treatment of infantile eczema resulting from BCG inoculation. Infantile eczema is the only allergic manifestation that is not due to the simultaneous presence of allergens and antibodies. The pathogenic factor of this type of eczema is the antibody, since when the antibody is neutralized by injection of a specific allergen it disappears from the blood within a few hours. This treatment produces a shock, however, which is not free from danger. The author prefers exerting an inhibiting action on the antibody. All methods of desensitization may be used. Professor Debré and his associates used cortisone with very good results, but relapses have been numerous. It has also been found that, following measles or other infectious disease, eczema disappeared for two to three weeks; only with primary infections is the recovery from eczema complete. Voringer had the idea of treating eczema in nursing infants with BCG vaccination with large doses (0.03 to 0.05 mg.). The eczema of 82 of 100 nursing infants was thus cured. In 28 cases there has been a relapse, but the dermatosis was much less severe. The younger the child the greater the chance of recovery. The effect on other manifestations of allergy is less evident than on the eczema itself; the antibody has been found in the blood serum of some infants five months after the vaccination, and in five cases asthma occurred during the second stage of infancy. The risks of BCG vaccination are negligible, although sometimes an induration or abscess is noted.

Cortisone.—B. N. Halpern, B. Benacerraf, J. P. Duesberg, and M. Briot reported experiments on the protective effects of cortisone in hemorrhagic, traumatic, and toxic shocks on animals whose adrenal glands had been removed. They also reported studies on the influence of cortisone and of antihistaminics on experimental dermatitis in the guinea pig. The dermatitis was provoked by *p*-phenylenediamine.

Rosacea of the Face.—R. Aron-Brunetière, J. Bourgeois-Gavardin, and J. Cohen reported the results of investigations of results of bacteriological tests performed on patients with rosacea of the face. They performed intradermal tests by injections of 0.1 cc. of a 1:100 dilution of various vaccines in the forearm of 115 patients. The results recorded 48 hours later showed three groups with different reactions. In the first group injections of enterococcus vaccine yielded negative results in 83.5% of cases, and in the second the results obtained with Staphylococcus, Streptococcus, Escherichia coli, and Proteus vaccines were roughly equal. On the other hand, in the third group tests were positive in 66% of cases with Eberth's bacillus (Salmonella typhosa), in 77.4% with S. enteridis, in 80.8% with S. paratyphi, and in 81.7% with S. typhi murium. The therapeutic results were interesting. Of a total of 105 patients, 80 received one or more courses of 3 to 9 gm. of chloramphenicol during five to seven days, 10 received aureomycin in the same dosage, and 15 were treated with a conbination of the two antibiotics. Results were as follows: 9 patients recovered completely, 5 were not improved, and several had more or less noticeable improvement. There were numerous relapses but they were less severe than the original condition and yielded to another course of antibiotics. The authors are considering the possibility of a focus of infection in this condition. According to this concept, bacteria would provoke disorders not as a result of their pathogenicity in a morbid focus but as a result of their antigenic property. Moreover, R. Aron-Brunetière, J. Cohen, and G. Melky reported on the excellent results they obtained in six cases of dyshidrosic eczema treated with chloramphenicol.

Uterine Fibromas and Corticotropin.—F. Hanon and J. Varnier (Baudelocque Hospital) have just evolved a new method of treatment of uterine fibromas with corticotropin (ACTH), the first results of which they have presented to the Paris Society for Gynecology and Obstetrics. They based their work on the fact that corticotropin possesses an anticollagenic and antifibrous action and on the conception that delivery is a particular stress provoking the production of corticotropin by the hypophysis. This would explain the involution of the uterus and its reduction to normal size within the six weeks following delivery. It is also a fact that fibromas appear to be smaller after delivery than before gestation. Laboratory investigations have shown a particular power of the blood of delivered women: cultures of uterine tissues from the female cat and the female rat subjected to the action of serum of pregnant, laboring, and postpartum women revealed that the first is capable of replacing the embryonal fluid of chickens, frequently used for the culture of tissues, and that it has an especially marked action on uterine tissue. On the contrary, the blood of women in the stage of postpartum involution does not favor any more the proliferation but is more likely to exert an inhibiting action. Clinically, repeated injection of a woman with a large uterine fibroma with blood taken during the postpartum period brought about the diminution of the fibroma by about one quarter.

Hanon and Varnier, attributing these experimental and clinical facts to the action of the obstetric stress on the blood, were induced to try corticotropin itself against uterine fibroma. For the first trials, the authors selected very large fibromas in order to avoid the errors of estimation of volume based on tracing of the fibroma, measurements, etc. The women had not had their menopause, their fibroma behaved as if it were a stable tumor, without congestive outbreaks inducing important functional and vesicular disorders, and in some cases male hormones had been administered without much success.

Every treatment was preceded by a Thorn test. Corticotropin was administered three times a week by deep intramuscular injections in a dose of 25 units and sometimes of 50 units for women with a particularly favorable Thorn's test and whose uterus had not decreased in size rapidly enough. In the average patient and in order to obtain lasting results the treatment was applied during one month and a half, followed by another identical series after an interval of one month; the maximum number of injections has been 45.

In all cases the diminution of volume of the fibroma has been considerable, and has been as much as decrease from the size of a head to that of a small tangerine. The height of the uterus passed in one case from 20 cm. to 9.5 cm. Sometimes the diminution was spectacular after three injections. Old vesical disorders disappeared, and at times a few injections induced pollakiuria. The authors noted disappearance of asthenia and very noticeable euphoria without any depressive psychosis following cessation of the treatment. Treatment with such small doses provoked no humoral modification; there were no signs of hypocalcemia, retention of chlorides, or glucide disorders. Arterial blood pressure was not increased; in a hypertensive patient it fell from 180/110 to 120/90 and returned to its initial level after cessation of treatment. The weight of patients who were kept on a salt-free diet did not vary more than 2 kg. No accident was noted; the very deep intramuscular injection avoided the formation of abscess. For technical reasons, intravenous perfusion could not be used in these cases although it is more effective with smaller doses. The very interesting results of this new method have been maintained for eight months at the time of writing. The authors are pursuing their investigations on a large scale.

Tuberculous Meningitis in Children.—On Oct. 24, 1952, Prof. R. Debré, H. E. Brissaud, H. Noufflard, and N. Naveau presented their results of treatment at the Medical Society of the Paris Hospitals. They used isoniazid in all cases, when Koch's bacillus was, or became, resistant to streptomycin. In these cases isoniazid transformed the prognostic of tuberculous meningitis. With the adjunct of *p*-aminosalicylic acid to isoniazid, patients with hopeless cases lived beyond the usually expected time. In five cases a survival was obtained notwithstanding the presence of streptomycin-resistant bacilli in vitro, in two cases the strains had a resistance exceeding 100 γ, i. e., were from 200 to 800 times stronger than that of strain H 37 RV. The authors also reported the results observed in three groups of children with tuberculous meningitis and treated with antibiotics since 1947. The first group consisted of 93 children treated since 1947 with short and discontinued administration of streptomycin: 40 died during the first 3

months and 27 have been living for 5 years without relapse. The second group consisted of 309 children treated with streptomycin between 1947 and 1952: 83 died after three months and 185 have been living for one to five years. In the third group of 15 children, 4 of whom were in a very serious condition, treated with isoniazid, only 1 died during the first three months. As to the recent technique of treatment, the authors emphasize the necessity of associating isoniazid with streptomycin and *p*-aminosalicyclic acid. Laboratory experiments revealed the existence of a synergia, or, at least, of an additional effect in the action of different antituberculous antibiotics. It is also necessary to maintain the strongest possible concentration of the antibiotic in the neighborhood of the lesions. The 150 cases of therapy performed have resulted in this conclusion. The authors found variations of the ratio existing between the levels in the cerebrospinal fluid and those existing in the blood. To the contrary of streptomycin the permeability of the meningeal barrier to isoniazid is always appreciable, even in healthy meninges. With isoniazid the variations fluctuate between 1 and 0.5, whereas with streptomycin they vary from 0.5 to 0; this means that the streptomycin level becomes null when the meninx is somewhat inflamed and the albumin level low. With isoniazid, and under similar conditions, the cerebrospinal fluid level is still half of the maximum found in the blood. Moreover, the diffusion of isoniazid administered in intrarachidian injections is remarkable. Half an hour after the intrarachidian introduction of 20,000 γ of this drug, less than 200 γ per cubic centimeter was found in the lumbar fornix but already 8 γ in the ventricle; two hours later, there was less than 100 γ in the fornix and 12 γ in the ventricle. The general plan of treatment consists in giving intravenously 0.5 to 1 gm. of streptomycin each 24 hours, orally 30 to 50 cg. per kilogram of body weight and 5 to 15 mg. of isoniazid per kilogram of body weight, and finally a daily intrarachidian injection of a mixture containing 20 to 30 mg. of streptomycin, 10 to 20 mg. of isoniazid, and 25 mg. of *p*-aminosalicylic acid. Intrarachidian injections were well tolerated by patients, and the 4,275 injections that were given provoked no accident.

SWEDEN

Improvement of Hospital Services.—Dr. Gunnar Berg of Stocksund is a member of a committee charged with the study of hospital administration in the county of Stockholm. His experience in hospital administration in Sweden has been supplemented by a study, during the summer of 1952, of the same problem in England. In *Svenska Läkartidningen* for Nov. 28, 1952, he presented a summary of his observations and some of his conclusions regarding reforms, the need for which is evident merely from a glance at hospital budgets. In the last decade the cost of hospital administration has more than trebled. At present it costs about 50,000 kronor per hospital bed in a building program, and about 15,000 kronor are spent per year on administration for each bed. In other words, running a hospital for three years costs approximately the same as building such a hospital. It is therefore necessary, before clamoring for new hospital construction, to make sure that each bed is properly used and that a shortage of personnel does not lead to the closing of hospital departments. For many years to come there will be a shortage of well-qualified hospital personnel. While some Swedish hospitals run ambulatory or outpatient departments where patients can be treated before they become inpatients, other hospitals lack such an auxiliary service, making them more expensive to operate.

The Swedish Ministry of Health has compiled statistics concerning the cost of administration of hospitals of various types in 1951. Between 1940 and 1951 there was a considerable shortening in the average duration of hospital medical and surgical treatment, but while in some provincial hospitals this shortening has been considerable it has hardly been noticeable in some of the hospitals in Stockholm, where the average duration of hospital treatment per patient was twice as long as elsewhere. These wide variations in the duration of hospital treatment may be interpreted as a hopeful sign, for they suggest that residence in hospital can be considerably shortened without impairment of efficiency.

Dr. Berg is critical of the present system of closing down hospital departments during the summer vacations and running the other departments at the same time with inefficient, locum tenens personnel. Would it not be wiser, he suggests, to spread vacations over a longer period as is done by the postal, telegraphic, and railway services? He would like to see hospitals so well staffed that it would not be necessary to engage incompetent outsiders for vacation services. He concludes that the effectiveness of hospitals will be greatly increased when the patients admitted to them are strictly in need of them and are not recruited from the ranks of the chronically ill, the aged, and convalescents, as well as those patients sometimes kept in hospital because of social difficulties rather than actual disease.

In a more recent publication Dr. Berg refers to a census taken on June 17-19, 1952, of all the hospital beds of the county of Stockholm. Thanks to the cooperation of the hospital staffs, exact information was obtained about the occupants of these beds. It was found that only 562 of the 667 occupants were bona fide patients requiring first class hospital treatment. While 84% were in hospital for good reasons, the remaining 16% should not have encumbered costly hospital beds. A considerable proportion (13%) of the hospital beds were not in use at the time of the census, yet at the same time in several hospitals there were waiting lists. A study of waiting lists showed that several of the patients on them were not suitable candidates for hospital treatment. Dr. Berg concludes that hospital administration can be made more effective by dismissing the chronically ill and the aged, particularly from the medical departments of hospitals in which they are found more frequently than in the surgical department. He also insists on raising the quality rather than the quantity of hospital personnel.

Deaths of Dr. Ahlbom, Dr. Lindsjö, and Dr. Tengwall.—The deaths of three internationally famous Swedish doctors have recently been announced. Prof. Hugo Ahlbom, born in 1900, died on Nov. 19, 1952, of chronic nephritis. His illness had, in recent years, interfered with his work at the Karolinska Institute, to which he was attached as professor of radiotherapy. Many years ago, Professor Ahlbom had qualified by the practice of surgery in a hospital for this high appointment and as a senior medical officer attached to Radiumhemmet. He was graduated as doctor of medicine in 1935 and was appointed docent in 1937. In 1944, he was put in charge of the department of radiotherapy at the Södersjukhus (a large hospital in Stockholm), and on July 1, 1950, he was appointed professor. Though only 52 years of age at the time of his death, he had made numerous important contributions to the literature of his speciality. In 1941 he published a study of 1,815 patients with cancer of the breast treated in the period 1921-1935 and given the benefits of preoperative and postoperative radiotherapy. A generous tribute to his work and to his many qualities is paid by Prof. Elis Berven of Stockholm in *Nordisk Medicin* for Jan. 16, 1953.

Dr. David Lindsjö, who died on Oct. 20, 1952, was well known as the senior school medical officer of Stockholm when, on the outbreak World War II in 1939, he was put in charge of the Swedish army medical service. During this time of crisis an efficient administrator was badly needed. The reforms he introduced into this service included provision for surgical hospital experience for medical students called to military service. Differences in policy led to his retirement in September, 1943, and to his return to his medical school duties in Stockholm, but he was recalled to his post in the army in 1944. A myocardial infarct in April, 1951, preceded a year earlier by signs of heart disease, did much to disable him. Later infarcts were followed by an intestinal infection with uremia and virus pneumonia that became chronic.

Dr. Ernst Tengwall, who was born in 1873 and who died on Sept. 29, 1952, was a general surgeon in charge of a large hospital in Hälsingborg for many years. Shortly before his death, he had been operated on for cancer of the colon. Complications developed, and he died a fortnight after the operation. Many of his publications dealt with the surgery of the prostate about which he was the highest authority in Sweden.

CORRESPONDENCE

DIAPHRAGMATIC HERNIA

To the Editor:—The article "Right-Sided Traumatic Diaphragmatic Hernia Simulating a Pleural Effusion," by S. M. Unger, M.D., in the Feb. 28, 1953, issue of THE JOURNAL, page 734, reminds me of a similar situation. This patient was discharged from the service in 1945 because of "far advanced active pulmonary tuberculosis." Apparently the only symptom he displayed was moderate dyspnea on slight exertion (he weighed 250 lb. [113.4 kg.]). The diagnosis was predicated solely on chest x-ray findings. The pulmonary diagnosis was reaffirmed several months later by the findings of another roentgenologist. A year later, a third roentgenologist stated that a roentgenogram of the chest showed that the heart, aorta, and mediastinum were displaced to the right. The right diaphragm was smooth and rounded. The right lung was normal. On the left side of the chest there was marked thickening of the pleura over the left lower lobe from the anterior third rib downward. There were multiple annular shadows but no evidence of fluid levels in this region. These findings were suggestive of old thickening of the pleura with fibrocaseous pulmonary tuberculosis involving the left lower lobe.

When I saw this patient, careful auscultation of the chest revealed occasional but unquestionable borborygmi. It was learned through questioning that the patient had had chest trauma two years before he was first seen by me. Predicated on the auscultatory findings and the patient's history, a diagnosis of left diaphragmatic hernia was made and a series of gastrointestinal studies was requested. The report (made by the third roentgenologist mentioned above) stated that the annular shadows seen on the left side of the thorax were due to collections of gas in the gastrointestinal tract and that the generalized thickening throughout the same region was due to a diaphragmatic hernia on the left that included the greater portion of the stomach, several coils of small bowel, and a considerable portion of the transverse colon.

It has been said repeatedly, but apparently not sufficiently, that careful physical examination still remains a vital and basic element in attaining proper diagnosis; roentgenologic examination alone may prove to be completely misleading. "Abnormal" laboratory and roentgenologic findings that are at complete variance with the dictates of common sense and clinical acumen should be viewed with considerable suspicion.

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VISITORS TO GREAT BRITAIN

To the Editor:—It may be of interest to some of the readers of THE JOURNAL who intend visiting Great Britain to know of the International Medical Visitors' Bureau, which was set up in 1950 by the Council of the British Medical Association to help medical practitioners from overseas during their stay in this country. Information is available on postgraduate education facilities, and visits to hospitals and clinics can be arranged. Assistance can be given in finding suitable accommodations, and general information on such matters as food rationing, traveling, and theaters is available.

The bureau can be of most service if the visitor gives as long notice as possible of his intended visit to Great Britain. Information on the following points would also be useful: projected date of arrival, mode of travel, period of stay, main objects of visit, and requirements from the bureau. The overseas visitor is cordially invited to visit the bureau at the address given below and talk over with me any points on which he may need advice or assistance.

H. A. SANDIFORD, M.C.
Medical Director, International
Medical Visitors' Bureau,
B.M.A. House, Tavistock Square,
London, W.C.1.

BLOOD TRANSFUSION

To the Editor:—The article "Use and Abuse of Blood Transfusion," by Bernard Straus and Jose M. Torres, in THE JOURNAL, Feb. 28, page 699, calls attention to many of the problems involved in the inconsidered use of blood transfusion; however, one conclusion reached by the authors seems unjustified. They state, "Of the total number of units, 27.3% was returned, indicating that insufficient thought was devoted to the ordering of blood." I think it is unavoidable, when calculating the blood needed at operation, in prolonged gastrointestinal bleeding and in various hemorrhagic states, that a fair amount of blood ordered on a stand-by basis will not ultimately be needed and will be returned to the hospital blood bank unused. The return of 27.3% does not seem excessive.

The authors state that a figure of 10 to 15% blood returns is reasonable. That figure is based on the experience of the English Ministry of Health and specifically applies to blood returned "by an average-size blood bank to the regional transfusion centers." This is entirely different from the drawing of blood from a hospital bank.

The careful and deliberate overestimation of blood needs does not indicate "that insufficient thought was devoted to the ordering of blood," but, on the contrary, that careful attention was paid to possible future demands in unpredictable situations. It seems wiser to draw more blood than necessary from the bank than to risk delays by underestimating possible needs. It is important, however, that unused blood be returned promptly to the bank and not allowed to become outdated.

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CHOICE OF BLOOD DONORS

To the Editor:—I have read with a great deal of interest the article "The Use and Abuse of Blood Transfusions" by Drs. Straus and Torres in the Feb. 28, 1953, issue of THE JOURNAL. I was particularly struck by the authors' comment on page 701. In 30 years' experience in blood transfusions, I have continually called attention to not only the blood type of the donor but also the type of the blood donor himself. I agree wholeheartedly, from recent experiences, with the statement of these authors that too often little attention is paid to the selection of donors. It has been our experience that careful selection of donors from friends and relatives of recipients lessens reactions and infections after blood transfusions.

At Inter-County Blood Banks, Inc., we believe that placing the responsibility for supplying blood replacement donors on the recipient is the safest means of obtaining blood.

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LEVIN TUBE FOR GALLBLADDER OPERATIONS

To the Editor:—Before or even during a gallbladder operation, insertion of a Levin tube through the nose into the stomach and leaving it in position with mild continuous suction for 24 hours after operation has been of great value. This procedure seems to prevent postoperative gastric dilatation. It also seems to reduce to a minimum nausea and intestinal distention. I have used this procedure for more than seven years and have noticed no ill effects. In several hospitals, the insertion of the Levin tube preoperatively has become routine. The procedure also keeps the stomach empty during operations by reducing to a minimum trauma to the stomach and duodenum caused by packing with gauze to obtain a better exposure.

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MISCELLANY

INFLUENZA TODAY

The purpose of this article is to give an account of this year's influenza epidemic up to the end of January, based on confirmed reports. First it is necessary to consider some technical facts. Three main types of the virus of influenza have so far been discovered; the two most important of them—A and B—comprise several sub-groups. From time to time new strains appear which may be quite different from previously known strains both as far as protection by vaccination is concerned and also in their ability to cause epidemics. The consequences are that epidemiological reports can only be correctly interpreted in terms of laboratory studies of the viruses responsible; that successful vaccination against influenza depends on knowledge of the virus causing the epidemic; and that continuous vigilance is necessary to detect new and potentially dangerous strains at the earliest moment.

In 1947 the Interim Commission of the World Health Organization initiated a network of influenza laboratories by setting up the World Influenza Center in London for the study of strains of virus isolated in different parts of the world. This was a logical development of a similar network previously set up by the Armed Services Commission on Influenza in the United States of America. A second reference laboratory in New York, known as the Strain Study Center for the Americas, is co-ordinating the work in the Americas. The world network now includes 55 laboratories in 44 different countries. Many more laboratories are co-operating informally.

The work of these laboratories continues summer and winter; for new strains may appear during the summer, and the strains causing epidemics during the winter in the southern hemisphere may spread to the northern hemisphere to cause outbreaks there a few months later. This apparently occurred during the 1950/51 outbreak when a strain of A-prime virus isolated in South Africa and Australia during June was found to be identical with one causing a severe outbreak in Liverpool in the following January. This became known as the Liverpool subtype; another simultaneously present in western Europe was called the Scandinavian subtype.

In 1952, laboratories in the WHO network again isolated an A-prime strain (Liverpool subtype) in South Africa in May, and the similarity of the circumstances to those of 1950 caused the World Influenza Center to issue in June a warning that there might be an outbreak due to this strain this winter.

In September 1952 viruses closely related to the 1951 A-prime (Liverpool) strains were isolated in Bahrein. Influenza was next reported in Tokyo in early December and viruses isolated both there and at Osaki were A-prime strains similar to those isolated during the 1950/51 outbreak and one has since been identified as the Scandinavian type.

In the latter half of December influenza was reported in the United States in the State of Missouri, the virus being similar to Scandinavian 1951 A-prime virus. Numerous reports from other states followed, indicating that the outbreak was widespread. Numerous isolations of virus throughout the country confirmed that an A-prime virus was responsible for the outbreak, although, as is frequently the case, a few sporadic cases of Influenza B were detected. The epidemic continued to increase during the first three weeks of January, with a small increase in the number of deaths attributed to influenza and pneumonia. Towards the end of January there were indications that in some states at least the epidemic had reached its peak.

In Europe, apart from a localized outbreak of Influenza B in Denmark in December, epidemic influenza appeared in France and Germany in early January, in southern England in the second week of January, followed by spread during the latter half of January both within these countries and north to the Netherlands and Denmark, and south to western Austria and Switzerland. Southern Europe, Spain, Italy and Yugoslavia were not involved at the end of January, nor was the north coast of Africa. Belgium, Ireland, Scotland and Finland had not been affected at this time but the disease had reached Iceland on January 30. In Sweden there were small epidemics in the western and southern parts; these were, however, apparently due to Virus B. The epidemic A-prime strain had not been detected by the end of January.

The epidemic in France increased markedly in the second week of January, flaring up almost simultaneously in Paris and in other parts of the country. The disease was mild. In the Paris region an A-prime virus was isolated and identified as closely resembling the 1951 Liverpool type. By the end of January it seemed probable that the epidemic had reached its peak. In Switzerland two strains of virus have been identified as the Liverpool type.

In Western Germany the outbreak appears to have begun along the southern reaches of the Rhine and to have spread north reaching Cuxhaven in the third week of January and Bremen by the end of the month. The epidemic also spread across the country. The epidemic had reached Western Berlin by the third week of January. In all areas the disease was mild and serological tests indicated that an A virus was responsible.

In the United Kingdom a mild form of influenza spread across southern England and Wales in the first half of January, and began to appear as localized outbreaks in the midlands and north in the third and fourth weeks. The epidemic had not reached Scotland by the end of the month. Several strains of virus have been identified as A-prime resembling the 1951 Scandinavian type and generally similar to the strains isolated in the U. S. A. but slightly different from the 1953 strains from Paris and Switzerland.

In Japan the epidemic which had affected mainly the north-eastern part of the country spread to the southwestern part in the middle of January. The responsible virus was identified as resembling the 1951 Scandinavian type.

Outbreaks were also reported beginning in December in various parts of the Pacific region including the Philippines and Hawaii where the virus was identified as A-prime. The true interpretation of the epidemiological data must await the results of further careful studies and comparisons of the viruses isolated in various parts of the world. However, it is clear that the viruses responsible for this epidemic are of the A-prime type and closely related to strains isolated in 1950/51.

Vaccination against influenza produces neither as lasting nor as complete an immunity as does, for example, vaccination against smallpox. In controlled trials in the past the unprotected individuals have been attacked about four times as often as the inoculated, but a number of the latter still become ill with the disease. It must be stressed that the vaccines at present (March 7, 1953) under test lend no hope of being able to eliminate the disease; the best that can be hoped for is to limit the effects of an epidemic by reducing the number of people who fall sick. A great deal remains to be learned about the best methods of producing, testing and using vaccines.

The use of influenza vaccines presents a number of problems. It is clearly impracticable to vaccinate the whole population each year. The questions are whom to inoculate and when. The answers will depend a great deal on local conditions and economy. It is hoped that some guidance on this will be forthcoming as a result of the trials at present in progress.

An important function of all health authorities is the protection of the public from dangerous, inferior or inactive products. To effect this it is necessary to lay down tests and standards to which products must conform. This has been done in some countries. There is good reason to believe that influenza vaccines will play a principal role in lessening the effects of influenza epidemics in the future. However, they give no hope of eliminating influenza altogether. A great deal of research work is in progress, and it may be hoped that solutions to many problems will be found in the not-far-distant future.

Abstract of a recent article by A. M. M. Payne, M.D., of the Division of Communicable Disease Services of the World Health Organization.

MEDICAL LITERATURE ABSTRACTS

UNITED STATES

A. M. A. Am. J. Diseases Children, Chicago

85:1-134 (Jan.) 1953

Skeletal Maturational Progress of Children with Chronic Nutritive Failure: Effect of Dietary Supplement of Reconstituted Milk Solids. T. D. Spies, S. Dreizen, R. M. Snodgrasse and others.—p. 1.

Prolonged Obstructive Jaundice in Infancy: III. Liver Function Tests. D. Yi-Yung Hsia and S. S. Gellis.—p. 13.

Crossed Laterality in Children: Report of Pediatric, Psychiatric, and Psychological Aspects of Three Cases. M. Seldowitz and A. B. Berman. —p. 20.

*Coccidioidomycosis of Bone in Children. J. Dykes, J. K. Segesman and J. W. Birsner.—p. 34.

Hydranencephaly. J. T. Olive and J. W. DuShane.—p. 43.

Perforation of Stomach in Newborn: Report of Two Cases with Necropsy Findings. W. W. Greene and D. F. Gose.—p. 47.

Coccidioidomycosis of Bone in Children.—Dykes and associates deplore the prevalence of misinformation regarding the morbidity and mortality of coccidioidomycosis. In their opinion, it is a chronic self-limiting disease that rarely ends fatally unless there is generalized systemic involvement in addition to the osseous involvement. They review data on 26 children with osseous coccidioidomycosis from the files of Kern General Hospital, Bakersfield, Calif. In all cases diagnosis was confirmed by skin tests, serologic studies, smears, cultures, and/or autopsy findings. The lesion is osteolytic in type, similar to that found in adults, and may occur as a single lesion or multiple lesions. It may or may not be accompanied by a pulmonary lesion or a cutaneous abscess. As a rule, the process is chronic, with a relatively good prognosis for eventual recovery. Parenchymal infiltration of the lung frequently precedes the bone lesion, and at the time of osseous coccidioidomycosis there may be only residual pulmonary adenopathy. In endemic areas the lesion is not uncommon, and elsewhere it may occur frequently enough that coccidioidomycosis should be considered when there are osteolytic lesions in children. An appropriate history of travel and previous residence may suggest this diagnosis.

A. M. A. Arch. Pathology, Chicago

55:1-84 (Jan.) 1953

Dissecting Aneurysms of Aorta in Persons Under 40 Years of Age. I. Gore.—p. 1.

Pathology of Pneumotropic Virus recovered from C3H Mice Carrying Bittner Milk Agent. E. R. Fisher and L. Kilham.—p. 14.

Histochemical Studies on Lens Following Radiation Injury. S. Permutt and F. B. Johnson.—p. 20.

Postmortem X-Ray Studies of Congenital Malformations of Heart. R. M. Collister, J. Dankmeijer, H. A. Snellen and W. H. van der Wel.—p. 31.

Hypervitaminosis A in Young Ducks: Epiphyseal Cartilages. S. B. Wolbach and D. M. Hegsted.—p. 47.

Nuclear Changes in Response to Continuous Irradiation. M. S. Burstone. —p. 55.

Diagnostic Significance of Fatty Cysts in Cirrhosis. W. S. Hartroft.—p. 63.

Elastin Content of Albuminoid Fraction of Human Aortae. D. M. Kraemer and H. Miller.—p. 70.

Vasal Component in Syndrome of Foix and Alajouanine: "Subacute Necrotizing Myelitis." K. T. Neuburger, C. G. Freed and J. Denst. —p. 73.

Periodicals on file in the Library of the American Medical Association may be borrowed by members of the Association or its student organization and by individual subscribers, provided they reside in continental United States or Canada. Requests for periodicals should be addressed "Library, American Medical Association." Periodical files cover only the last 11 years, and no photoduplication services are available. No charge is made to members, but the fee for other borrowers is 15 cents in stamps for each item. Only three periodicals may be borrowed at one time, and they must not be kept longer than five days. Periodicals published by the American Medical Association are not available for lending but can be supplied on purchase order. Reprints as a rule are the property of authors and can be obtained for permanent possession only from them.

Titles marked with an asterisk (*) are abstracted.

A. M. A. Arch. Surgery, Chicago

66:1-128 (Jan.) 1953

Observations on Effect of Methantheline (Banthine®) Bromide in Urological Disturbances. J. Lapides and A. I. Dodson Jr.—p. 1.

Gastrointestinal Hemorrhage from Benign Tumors of Duodenum. J. D. Briggs and J. F. Roberts.—p. 10.

Variational Anatomy of the Hepatic, Cystic, and Retroduodenal Arteries: Statistical Analysis of Their Origin, Distribution, and Relations to Biliary Ducts in 200 Bodies. N. A. Michels.—p. 20.

Evaluation of Trichloroethylene as an Anesthetic and Analgesic Agent. W. K. Nowill, C. R. Stephen and P. W. Searles.—p. 35.

New Technique of Surgical Management of Patent Ductus Arteriosus. H. A. Davis.—p. 48.

Diaphragm Pump for Artificial Circulation, Tyson-Bowman Pump. J. L. Southworth, E. C. Pierce II., T. Tyson and R. L. Bowman.—p. 53.

*Subacute and Chronic Osteomyelitis: Treatment with Use of Chemotherapeutic Agents, Antibiotics, and Primary Closure; Follow-Up Report. F. D. Dickson, R. L. Diveley and R. H. Kiene.—p. 60.

Surgical Management of Shoulder Girdle Syndromes: New Operative Procedure of Hyperabduction, Costoclavicular, Cervical Rib, and Scalenus Anticus Syndromes. J. W. Lord Jr.—p. 69.

New Concept of Vesical Innervation: Relationship of Vesical Innervation of Bladder Management Following Abdominoperineal Proctosigmoidectomy. L. E. McCrea and D. L. Kimmel.—p. 84.

*Relationship Between Benign Breast Disease and Cancer. E. F. Lewison and J. G. Lyons Jr.—p. 94.

Midthigh Amputations for Arteriosclerotic and Diabetic Gangrene: Analysis of 113 Consecutive Amputations in 108 Patients, 1939 to 1952. L. Carp.—p. 115.

Treatment of Osteomyelitis.—In the past four types of surgical treatment of osteomyelitis have been widely used: (1) débridement, followed by repeated packing to provide drainage; (2) the maggot method of Baer; (3) the Dakin-Carrel method, and (4) the Orr method. Dickson and associates believe that superior results can be achieved by means of débridement and primary wound closure supplemented by the use of antibacterial agents. They report on results of 140 such operations in 104 patients with hematogenous osteomyelitis. Their procedure consisted of application of a tourniquet to the extremity; injection of cutaneous sinus tracts with methylene blue to stain and outline all necrotic bone tissue; removal of all dead tissue with chisel and gouge; saucerization of the cavity and smoothing of the edges with a thick burr; excision of all scar tissue to permit healthy tissue to fill in the bone cavity; thorough irrigation of wound with isotonic sodium chloride, and introduction of 0.5 to 1.0 gm. of sulfathiazole powder into the wound with a nasal insufflator. The deep soft tissues, including mobilized normal muscle if necessary, were then brought into close contact with the denuded area of bone by means of interrupted sutures. Additional sulfathiazole powder was applied over this layer and the superficial structures were closed. A dressing was applied in such a manner as to press the soft parts into the bone cavity. The extremity was mobilized in a plaster cast for three weeks. Systemically the patients were treated with sulfathiazole, sulfadiazine, penicillin, aureomycin, streptomycin, oxytetracycline, ("terramycin") or chloramphenicol, but the last four antibiotics were not used in sufficient number of cases, or the patients were not followed long enough to warrant any conclusions. Penicillin (400,000 units) was given every four hours on the day preceding operation and was continued for 12 days thereafter. When penicillin was used systemically and sulfathiazole locally, the incidence of primary healing was definitely higher than when sulfadiazine or sulfathiazole was used systemically and sulfathiazole locally. The antibacterial agent used systemically should be adapted to the sensitivity of the predominating organism but if this cannot be determined, the use of penicillin before and after operation is preferred. The administration of staphylococcus toxoid is a useful adjunct in the prevention of recurrences. In 117 or more than 83% of the 140 interventions healing took place by primary intention. The majority of the patients were discharged from the hospital in three weeks and were able to work in a month. The authors feel that the local use of sulfathiazole has been the factor securing primary healing, but only if a primary closure is carried out. Introduction of

sulfathiazole into the wound that is left open or only partially closed is probably useless. The advantage of using sulfathiazole is that its bacterial inhibiting action extends for a number of days.

Benign Breast Disease and Cancer.—Between 1925 and 1942, 451 patients with benign breast disease were operated upon at the Johns Hopkins Hospital. Of these, 85.4%, or 385 patients, were successfully followed over a 1 to 25 year survival span. The average follow-up period was 13.6 years, with 75% of the patients being followed from 10 to 25 years. The pathological diagnosis was fibroadenoma in 200 patients, chronic cystic mastitis in 153 patients, and papilloma in 32 patients. The maximum incidence of fibroadenoma was found in patients between 26 and 30 years of age, of chronic cystic mastitis in patients between the ages of 41 and 45, and of papilloma in patients between the ages 46 and 50. The over-all average age at onset of benign breast disease was 36 years. Simple excision was performed in 92%, or 352 patients. Benign breast disease recurred in 50 (13%) of 385 patients. Cancer of the breast developed in 7 (1.8%) of the 385 patients surveyed. The chances of acquiring breast cancer appeared to be 2.6 to 3.6 times greater in the present series than in the general female population in the same age range. The present study is in accord with current knowledge, which accepts a predisposition of patients with benign breast disease to breast cancer. Thus, all patients with benign breast disease should be regularly examined at periodic intervals.

American Journal of Medicine, New York

14:1-138 (Jan.) 1953

*Cardiac Catheterization in Interatrial Septal Defect. R. S. Cosby, G. C. Griffith, W. J. Zinn and others.—p. 4.

Precipitation by Pulmonary Infection of Acute Anosia, Cardiac Failure and Respiratory Acidosis in Chronic Pulmonary Disease: Pathogenesis and Treatment. D. J. Stone, A. Schwartz, W. Newman and others. —p. 14.

Pulmonary Function Studies in Bronchial Asthma. I. In Control State. J. A. Herschfus, E. Bresnick and M. S. Segal.—p. 23.

Id.: II. After Treatment. J. A. Herschfus, E. Bresnick and M. S. Segal. —p. 34.

Paradoxical Result Obtained with Hickey-Hare Test for Diabetes Insipidus: With a Note on Regulation of Chloride Excretion. J. M. Little, E. H. Yount and W. M. Kelsey.—p. 41.

*Temporal Arteritis: Critical Evaluation of This Disorder and Report of Three Cases. J. K. Meneely Jr. and N. H. Bigelow.—p. 46.

Effects and Treatment of Nerve Gas Poisoning. D. Grob and A. M. Harvey.—p. 52.

Mechanism of Blood Coagulation in Normal and Pathologic Conditions. M. Stefanini.—p. 64.

Cardiac Catheterization in Interatrial Septal Defects.—Cardiac catheterization was performed on 10 patients between the ages of 7 and 52 with proven interatrial septal defects, and in 7 additional patients, in whom the diagnosis of interatrial septal defect was "presumptive," since their findings may be simulated exactly by partial transposition of the pulmonary veins into the right ventricle. Of the 10 patients with proven interatrial septal defect, shortness of breath was conspicuous in all, and 4 of them had a history of cyanosis. Two distinct types could be distinguished among these patients. The younger patients without a history of cyanosis were shown to have high arterial oxygen saturations, high left atrial pressures, a pronounced rise of oxygen content between venae cavae and right atrium, full pulmonary vascular shadows and, frequently, a systolic pulmonic thrill. The older patients and those with other defects or heart failure had a history of cyanosis, low arterial oxygen saturation, elevated right atrial pressures, little or no rise of oxygen content between superior vena cava and right atrium, less prominent vascular shadows, and absence of a pulmonary thrill. An atrial septal defect can be positively identified only if the catheter passes through the defect. In the authors' series the most definitive approach to this problem has been to show the catheter positioned in a left pulmonary vein, and then successively withdrawn into the left atrium and right atrium. Other findings in cardiac catheterization are not wholly diagnostic. In all but one of the 10 patients, the catheter entered the left auricle. In four patients the pulmonary vein was entered as well. When an interatrial septal defect is the only anomaly, the findings depend in large measure on whether the shunt is predominantly from left to right or from right to left; the direction of the shunt is determined by the pressure gradient between the atriums during the cardiac cycle. The cause for cyanosis in atrial septal defects lies primarily in the presence of a right to left shunt, and all the authors' patients with the latter anomaly had cyanosis. A rise of oxygen content in the right atrium in comparison to the oxygen content of the superior vena cava is not necessarily present in proven atrial septal defects. Pulmonary vascular disease may contribute to the pulmonary resistance but is not a significant factor in the production of cyanosis.

Temporal Arteritis.—The occurrence of temporal arteritis, a condition described first in 1932 by Horton and others, in three patients between the ages of 67 and 71 is reported by the authors. One of the patients died of the disease and necropsy was performed. Temporal arteritis is characterized by severe headache, pain in the temporal region, and the presence of inflamed, thickened and tortuous temporal arteries. Constitutional symptoms of a generalized infection are also present. The characteristic pathological lesion consists of proliferation of inflamed fibrous tissue of all layers of the affected vessel, together with focal necrosis and granulomatous lesions associated with foreign body giant cell formation in the media. Good therapeutic results were obtained by procaine hydrochloride injection, surgical excision of a portion of the diseased vessel and, in at least one case, antihistamine treatment consisting of 50 mg. of diphenhydramine (benadryl®) hydrochloride given three times daily. The disorder is usually self-limited and nonfatal. Although temporal arteritis is generally a localized vascular disease of the temporal arteries, other cranial arteries, as well as arteries in other parts of the body, may show the same type of lesion. The authors believe the term temporal arteritis should be restricted to cases in which the affected vessel has undergone the characteristic granulomatous change associated with foreign body giant cells. They also suggest changing the regional designation temporal arteritis to that of a pathological entity, such as nonspecific granulomatous arteritis or, more simply, granulomatous arteritis.

American Journal of Surgery, New York

85:1-124 (Jan.) 1953

Dysgerminoma of the Ovary. R. F. Looker, W. P. Callahan Jr. and F. E. Barry.—p. 4.

Intestinal Obstruction in Advanced Ovarian Carcinoma. C. J. Schein and J. S. Krakauer.—p. 13.

Surgical Indications in Massive Hemorrhage from Peptic Ulcerations. L. Breidenbach, N. J. Saltz and H. Soroff.—p. 18.

Office Management of Leukorrhea. G. Blinick and S. A. Kaufman.—p. 27.

Value of Proctosigmoidoscopic Examination in Management of Intestinal Obstruction. R. H. Burnikel and H. C. Sprecher.—p. 35.

Carcinoid of the Rectum: Report of Three Cases. P. C. Morton and R. W. Fraser.—p. 43.

Mesenteric Vascular Occlusion. J. J. Carucci.—p. 47.

*Unfavorable Reaction to Use of Streptokinase-Streptodornase in Indolent Postroentgen Irradiation Ulceration. S. G. Castigliano and C. J. Rominger.—p. 55.

Some Aspects of Super Voltage Roentgen Therapy. F. W. O'Brien Jr. —p. 62.

Relationship of Gonadal Imbalance to Thyroid Disease. F. P. Ferrer and T. H. McGavack.—p. 67.

Non-Tropical Sprue-Like Syndrome Caused by Dilated Pouch Following Side-to-Side Anastomosis. H. Sneierson.—p. 71.

Transduodenal Approach in Surgery of Common Bile Duct. F. D. Conole.—p. 76.

Femoral Hernia. E. F. McLaughlin.—p. 79.

Bony Defects of Head Repaired with Cancellous Bone. G. K. Lewis. —p. 83.

Transplantation of Ureters into Isolated Ampulla of Rectum After Total Cystectomy. V. Levitsky.—p. 91.

Unfavorable Reaction to Streptokinase-Streptodornase in Irradiation Ulceration.—Castigliano and Rominger present two cases of late postirradiation ulceration of the anterior chest wall, which showed an extremely unfavorable and almost fatal reaction to treatment with streptokinase-streptodornase. The area of ulceration and the necrotic material increased with amazing rapidity. A febrile reaction developed which seemed due to increased absorption of pyogenic material as a result of digestion of the "walling off process." Perforation of the chest wall appeared likely, and mediastinal perforation was imminent. Both cases were complicated by an eczematoid type of dermatitis,

which occasionally accompanies indolent postirradiation ulceration. Heavy doses of ionizing irradiation produce tissue changes that impair blood supply and vitality. Many cells are just able to maintain their viability, under ideal conditions, but under even the mildest adversity, these marginal cells die. Late irradiation ulceration develops, either spontaneously or following trivial injury, and further impairs the vitality of many of these marginal cells by superimposed infection. Streptokinase-streptodornase appears to add to this adverse environment. Although these enzymes have no effect upon living cells, the authors feel that devitalized, heavily irradiated, marginally surviving cells are affected by these enzymes just as are necrotic cells, and are destroyed. In consequence the necrotic material increases in amount. With these thoughts and experiences in mind the authors discontinued the use of streptokinase-streptodornase in the management of late postirradiation ulcers. It appears dangerous to use this material in postirradiation ulcers in areas such as the chest wall where perforation may develop, or in areas where large blood vessels are contained in the tissue bed, for example, in the axilla, groin, or neck. It may prove to be useful in situations in which there is a deep tissue bed such as in the thigh or buttocks.

Am. J. Syphilis, Gonorrhea and Ven. Dis., St. Louis

37:1-100 (Jan.) 1953

Treponemal Immobilization Test in the United States Navy. R. A. Nelson Jr.—p. 1.

Production of Immobilizing Antibodies Unaccompanied by Active Immunity to Treponema Pallidum as Shown by Injecting Rabbits and Mice with Killed Organisms. C. P. McLeod and H. J. Magnuson.—p. 9.

Preliminary Study of Apparent Biological False Positive Reactions in Four Serological Tests for Syphilis, and Treponemal Immobilization Test. S. Olansky, A. Harris and J. H. Hill.—p. 23.

Treponema Pallidum Buds, Granules, and Cysts as Found in Human Syphilitic Chancres and Seen in Fixed Unstained Smears Observed Under Dark-Ground Illumination. W. E. Coutts and W. R. Coutts.—p. 29.

Mass Eradication Treatment of Treponemal Diseases with Penicillin, Laboratory and Clinical Basis for Selection of Effective Schedules. C. R. Rein and D. K. Kitchen.—p. 37.

Blood Testing for Syphilis in Island Population. J. S. Moorhead, G. S. Usher, J. F. O'Brien and others.—p. 46.

Studies on Granuloma Inguinale. I. Bacteriologic Behavior of Donovania Granulomatis. J. Goldberg, R. H. Weaver and H. Packer.—p. 60.

Id.: II. Complement Fixation Test in Diagnosis of Granuloma Inguinale. J. Goldberg, R. H. Weaver, H. Packer and W. G. Simpson.—p. 71.

Penicillin Alone and In Combination with Mercury Succinimide and Potassium Iodide in Treatment of Acute Syphilitic Orchitis of Rabbits. J. A. Kolmer.—p. 77.

Annals of Internal Medicine, Lancaster, Pa.

38:1-178 (Jan.) 1953

*Follow-Up Studies on First 1,000 Patients Operated on for Pulmonary Stenosis or Atresia (Results Up to March, 1952). H. B. Taussig and S. R. Bauersfeld.—p. 1.

Iatrogenic Heart Disease. H. B. Weinberg.—p. 9.

*Treatment of Angina Pectoris with Khellin. Part II. J. J. Conn, R. W. Kissane, R. A. Koon and T. E. Clark.—p. 23.

Spontaneous Adrenal Hemorrhage in Adult: Literature Review and Report of Two Cases. S. J. Berte.—p. 28.

Motility of Small Intestine During Emotional Reactions. H. P. Roth, R. N. Ferreri, M. A. Petti and M. W. Evans.—p. 38.

Far East Command Conference on Epidemic Hemorrhagic Fever: Introduction. J. H. McNinch.—p. 53.

Early Field Diagnosis of Epidemic Hemorrhagic Fever. W. F. Ganong, E. Zucker, C. K. Clawson and others.—p. 61.

Early Diagnosis of Epidemic Hemorrhagic Fever—Experiences in Forward Echelons of Medical Service. E. F. Counts and R. Seltser.—p. 67.

Gross Anatomic Features Found in 27 Autopsies of Epidemic Hemorrhagic Fever. W. H. Kessler.—p. 73.

Pathology of Epidemic Hemorrhagic Fever. R. L. Hullinghorst and A. Steer.—p. 77.

Clinical Aspects of Renal Phase of Epidemic Hemorrhagic Fever. W. E. Swift Jr.—p. 102.

Epidemic Hemorrhagic Fever: A Summarization. C. L. Leedham.—p. 106.

Follow-Up Studies on One Thousand Patients Operated on for Pulmonary Stenosis or Atresia.—The first 1,000 patients operated on for the alleviation of pulmonary stenosis or atresia by a systemic-pulmonic anastomosis, between November, 1944, and October, 1950, have been followed up to March 1, 1952. Thus all have been followed for at least 18 months and some for more than seven years. The preoperative diagnosis was tetralogy of Fallot in 857 patients, tricuspid atresia in 62, pseudotruncus arteriosus in 27, dextrocardia or dextrorotation in 32, situs inversus with levocardia in 11, and the remaining 11 patients were believed to suffer from lack of adequate pulmonary blood flow but the exact nature of the malformation was not certain. Both the immediate mortality rate and the mortality rate at the end of eight months are given, because if the malformation is such that the heart cannot adjust to the altered circulation, these patients either die from heart failure or show evidence of severe congestive failure within this period. In tetralogy of Fallot the immediate mortality was 15%; 14 additional patients died during the first 8 months following the operation so that the total mortality was 17%. Although 43 of 62 patients with tricuspid atresia were greatly improved by operation, and 6 obtained fair results, 21% died at or shortly after operation, and at the end of eight months the mortality rate was 26%. About the same mortality (death of one-fourth of the patients) occurred in those with truncus arteriosus, dextrocardia, or dextrorotation and in those with an uncertain diagnosis. In the 11 patients with situs inversus and levocardia the immediate results were good in all but 2 patients and these 2 had fair results, but eight months later 2 patients had died of congestive failure. The results of operation are better and the risk of operation is less for patients with tetralogy of Fallot than for any of the other malformations. The long-time results were analyzed according to whether the patient obtained a good or a fair result or was unimproved by operation. In the entire series there have been 53 late deaths, 43 in the group of patients with tetralogy of Fallot and 10 among those with atypical malformations. The mortality rate has been significantly lower among those who obtained a good result than in the other two groups. One hundred fourteen patients have failed to maintain improvement; 28 of these have had a second operation, and the others are still pending. During the seven year period since the first operation there have been only 22 cases of subacute bacterial endocarditis, with four deaths.

Treatment of Angina Pectoris with Visammin.—In a previous paper, Conn and associates had reported results obtained with visammin (khellin) in the form of ammivin.[9] The present communication is concerned with 42 patients with angina pectoris who were treated with a brand of visammin known as khelloyd.* Each patient was instructed to record the number and severity of anginal attacks and the number of glyceryl trinitrate (nitroglycerin) tablets taken daily. All patients except a few were observed during a control period in which they received placebos identical in appearance to the tablets containing the drug. They were then started on visammin and observed for varying periods up to one year. The patient took 25 mg. of visammin a day for the first week, and the dose was thereafter increased at weekly intervals by 25 mg. a day until a therapeutic response or a reaction occurred. In the event that the patient suffered undesirable side-effects, the dose was decreased. A parenteral solution containing 40 mg. of visammin per cubic centimeter was employed as adjunctive therapy in six patients. These patients received 18 intramuscular and 7 intravenous injections of 2 cc. each, given at semiweekly or weekly intervals. In half of the patients reactions in the form of nausea and vomiting were encountered, the reactions in 12 patients being severe enough to require cessation of therapy. Twenty-five of the 42 patients had a favorable response to the visammin, 2 patients had an unfavorable response, and 14 patients were unchanged. One patient died before the study was completed after showing marked initial improvement. These results are not essentially different from those obtained with the other visammin preparation, investigated earlier, and they are in general agreement with those reported by the majority of other investigators in this country. Visammin is of value in the treatment of the anginal syndrome, but will not replace the other vasodilators to any great extent unless a method of decreasing the incidence of side-effects is discovered. Since the first group of patients received coated tablets and the second group received pain tablets of visammin, the use of the coated tablet evidently

affords no particular advantage or disadvantage in regard to the clinical response or side-effects. In fact, there would appear to be little or no choice between the two brands of visammin employed in these studies.

Anesthesiology, Philadelphia

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Ether Hyperglycemia as Influenced by Premedication and Pentothal Induction. W. P. Bass, D. T. Watts and H. F. Chase.—p. 18.
Quantitative Evaluation of Cardiovascular-Stimulant Drugs in Barbiturate Depression of Heart of Dog. K. J. Boniface and J. M. Brown.—p. 23.
Tetraethylthiuram Disulfide (Antabuse®) and Anesthetic Agents. B. M. Cooper, H. C. Slocum and C. R. Allen.—p. 29.
*Continuous Epidural Block in Treatment of Pancreatitis. T. Walker and W. E. Pembleton—p. 33.
Analysis of Small Concentrations of Trichloroethylene Vapor by Interferometry. K. D. Hall, L. N. Garlington, W. K. Nowill and C. R. Stephen.—p. 38.
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Anesthesia for Mitral Commissurotomy. J. W. Pender.—p. 77.
The Negro as an Anesthetic Risk. M. F. Poe.—p. 85.

Continuous Epidural Block in Treatment of Pancreatitis.—Splanchnic anesthesia has been used in the treatment of pancreatitis in the belief that it would relax the sphincter of Oddi and allow drainage of the necrotizing and digestive fluid from the pancreas into the duodenum, and would dilate the spastic blood vessels in the involved region. Some investigators have reported complete relief from a single injection, but others have found that a single injection gives only temporary relief, and repeated injections are required for adequate treatment. To obtain blockage of the splanchnic nerves for a longer time, Walker and Pembleton used continuous epidural block by means of the catheter technique in 17 patients with a presumptive diagnosis of pancreatitis. Subsequently, it was demonstrated that two of the patients did not have pancreatitis. At autopsy, one of them was found to have a dissecting aneurysm of the abdominal aorta, and yet the epidural block gave complete relief for a few hours. The other patient, in whom the block had not given relief, died, and a carcinoma of the pancreas was disclosed at autopsy. Observations indicated that pancreatitis may be confused with other acute abdominal conditions and that the degree of elevation of the serum amylase content is inconsistent. The 15 patients who were believed to have had pancreatitis obtained relief of symptoms with the block. The continuous technique offers a simple means of repeating the blocks as long as necessary.

Connecticut State Medical Journal, Hartford

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Evaluation of Psychosomatic Medicine. E. M. Smolen.—p. 3.
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Delaware State Medical Journal, Wilmington

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Gastroenterology, Baltimore

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*Preliminary Study Concerning Possibility of Dietary Carcinogenesis. R. Willheim and A. C. Ivy.—p. 1.
Allergic Manifestations in Gastrointestinal Tract. A. F. R. Andresen.—p. 20.
Effect of Alcohol on Canine External Pancreatic Secretion. F. P. Brooks and J. F. Thomas.—p. 36.
*Study of Gastric Function in "Decorticate" Man with Gastric Fistula. R. K. Doig, S. Wolf and H. G. Wolff.—p. 40.
Endoscopic Aspects of Gastric Mucosa in Relation to Its Function: Part I. In Gastritides. S. J. Stempien, J. E. Oritt and N. W. Karr.—p. 45.
Id.: Part II. In Benign Gastroduodenal Ulcer. S. J. Stempien, J. E. Oritt and N. W. Karr.—p. 55.
Use of Anesthetic Agents in Gastroscopy. J. S. Atwater.—p. 60.
An Amebiasis Complement Fixation Test in Diagnosis of Intestinal Amebiasis. F. Steigmann, W. H. Shlaes, S. Mintzer and G. Schaefer.—p. 70.
Lipomatous Pseudohypertrophy of Pancreas. H. N. Robson and G. B. D. Scott.—p. 74.
Bulbar Defects in Pancreatic Neoplasm Resembling Duodenal Ulcer. C. Gottlieb, S. L. Beranbaum and A. M. Wald.—p. 82.

Dietary Carcinogenesis.—Twenty-seven dyes, many of them being certified food colors, were fed to rats in a concentration of about 4% of the diet by dry weight. It was observed that the dyes differentially stain the mucosa of the various parts of the alimentary tract. The glandular stomach was stained by these dyes more readily than the intestinal mucosa and the colonic mucosa, whereas the epithelium of the forestomach showed no coloration or only a very faint one with most dyes. The mucosa of the glandular stomach was found at necropsy to be grossly swollen in many instances. Microscopically edema was often present and the cells stained poorly. They were often vacuolated and showed a loss of cytoplasmic pyroninophilia. Round cell infiltration was sometimes seen, but the most striking and frequent microscopic finding was the presence of granular, black to brown-colored deposits within the cells of the stained mucosa. These consisted probably of condensed or conjugated and therefore less soluble states of the absorbed dyes. When the feeding of the dyes was discontinued for 24 hours, the staining disappeared. In the case of some dyes, two or three days were required to lose the stain. The absorbed dyes were eliminated at least partly in the secretions of the gastric mucosa. In seven of 33 animals fed amaranth, ponceau SX, light green S.F. Yellowish, Guinea green B, and hematoxylin respectively, malignant tumors of the lymphatic tissue (lymphosarcomas) have been observed after feeding the dyes for from 6 to 20 months. Fifty control animals observed for 20 months or more failed to develop tumors. The authors tentatively ascribe the relatively frequent occurrence of the tumors to a chronic "strain" on the lymphatic or the reticuloendothelial system, induced by the relatively enormous quantity of continuously absorbed dye substance. The feeding of three other dyes, namely, erythrosine, ponceau 3R and Sudan IV for from 12 to 19 months was in 5 of 16 animals associated with the occurrence of cirrhotic changes in the liver with numerous hyperplastic foci, resembling cholangiomas and in one case hepatoma, but without definite invasive characteristics. Hepatic cirrhotic changes were not observed in the 50 control animals.

Gastric Function in Decorticate Man.—Gastric function was studied in a 31-year-old man who sustained extensive injury to the cerebral cortex as a result of an automobile accident. This rendered him permanently unconscious. Shortly after his accident a tracheotomy and gastrostomy were performed to facilitate his care. The value of this patient as an experimental subject lay in the fact that his stomach was easily accessible to study through the gastrostomy opening to the left and below the umbilicus, and that he did not react on the basis of interpretation of events in his environment. Observations were made on the appearance of the gastric mucosa seen through a cystoscope, on motor activity recorded from an inlying balloon, and on secretion withdrawn at appropriate intervals. Much automatic activity was shown that corresponded closely to that of intact human beings. There was no response to intravenously administered insulin or to vestibular stimulation. The main difference was that the stomach did not react to stimuli requiring interpretation and involving the highest integrative functions. Thus there was no reaction to noxious stimulation and no conditioned reactions, and the effects of even small amounts of pharmacodynamic agents were remarkably uniform.

Geriatrics, Minneapolis

8:1-64 (Jan.) 1953. Partial Index

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Quinidine for Chronic Auricular Fibrillation in Patient Over 60. E. H. Yount, M. Rosenblum and R. L. McMillan.—p. 19.
Training of Geriatrician. M. W. Thewlis.—p. 23.
Management of Silent Staghorn Calculi in the Aged. A. C. Drummond. —p. 27.
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Indiana State Medical Assn. Journal, Indianapolis

46:1-88 (Jan.) 1953

*Cost Allocations of Overtreatment Dermatitis. L. E. Gaul and G. B. Underwood.—p. 17.
Carotid Sinus Reflex: II. Use of Normal Reflex in Bedside Diagnosis Treatment. D. L. Urschel.—p. 21.
Clinical Observations on Pancreatic Malignancies. T. S. Malinowski. —p. 25.

Overtreatment Dermatitis.—Convinced that overtreatment of patients is as great an evil as inadequate treatment, Gaul and Underwood studied the incidence of contact dermatitis due to over-treatment during a six weeks period. Patients with a diagnosis of contact dermatitis, and who had had previous topical therapy, were asked to bring in all the remedies they had used, and the sequence of application was recorded. The onset, etiology, and course of the dermatitis was then correlated with the sequence of topical therapy. When the acute stage of the dermatitis had passed, patch tests were performed with these remedies. As a rule the severity of the patch test reactions to the remedies followed closely the course of the dermatitis. Patch test reactions proved that 27% of contact dermatitides were produced by topical treatments. The histories of 16 over-treated patients showed the usual series of events. Wives or husbands, fathers or mothers, relatives and friends, and acquaintances, and lay diagnosticians, including hawkers, treat the skin. Some of the remedies producing over-treatment had been prescribed by physicians, which is proof that skin preparations, particularly antipruritics, are marketed with insufficient pharmacological study. The evil of over-treatment can be reduced by an educational campaign that prevents dermatitis, needless suffering, and catastrophic drains on the family budget. Teaching patients the need to search out the cause of itching, and that dermatological symptoms most commonly warn of injury to the skin, is a program that protects the health of patients and increases the stature of dermatology. Daubing cutaneous symptoms, masking their warning calls with antipruritic chemicals, is a frustrating endeavor.

Journal of Immunology, Baltimore

70:1-128 (Jan.) 1953

Simple Method for Obtaining Highly Potent Tetanus Toxin. W. Koch and D. Kaplan.—p. 1.
Standardization of Cobra (Naja Flava) Venom Using Graded Response Method. P. A. Christensen and D. P. Finney.—p. 7.
Production of Antibody by Patients with Acute Viral Hepatitis. P. L. Eighman, R. W. Miller and W. P. Havens Jr.—p. 21.
Evaluation of Prolonged Antibiotic Therapy in Mice with Chronic Brucella Infection Due to Brucella Melitensis. J. M. Shaffer, C. J. Kucera and W. W. Spink.—p. 31.
Relation Between Infectivity and Hemagglutinin of Murine Encephalomyelitis Virus. M. C. Morris.—p. 39.
Virus Growth in Tissue Culture Fibroblasts: I. Influenza A and Herpes Simplex Viruses. C. S. Stulberg and R. Schapira.—p. 51.
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Id.: II. Inhibition by Antibody, and Antibody Requirements of Reaction. F. L. Adler.—p. 79.
In Vitro Action of Mustards on Infective and Toxic Components of Influenza A (PR8) Virus. J. Fong and E. Bernal.—p. 89.
In Vitro Action of Sulfur Mustard and Chloroethylamine Derivatives Upon Antigenicity of Influenza A (PR8) Virus. J. Fong and E. Bernal. —p. 97.
Studies with Shigella Dysenteriae (Shiga): IV. Immunological Reactions in Monkeys to Toxins in Isolated Intestinal Pouches. S. E. Branham, G. M. Dack and D. B. Riggs.—p. 103.
Study of the Relation of Antibiotics, Vitamins and Hormones to Immunity to Infection. L. A. Terzian, N. Stahler and H. Miller.—p. 115.
Immunological Relationship Between Human and Bovine Serum Albumin. L. R. Melcher, S. P. Masoueredis and R. Reed.—p. 125.

Journal of International College of Surgeons, Chicago

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Plastic Reconstruction of Hip with Nylon. R. Charry.—p. 1.
Malignant Disease of Urinary Bladder. M. K. O'Heeron, R. Riddel, J. Bozzeil and others.—p. 25.
Prevention of Iatrogenic Trauma in Otolaryngology. F. L. Lederer.—p. 43.
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Medical Problems in Disease of Gallbladder. F. B. McGlone.—p. 78.
Surgical Management of Diverticulitis. D. H. Witte, D. W. Ovitt and J. D. Conway.—p. 87.
*Orthopedic Treatment of Acute and Subacute Poliomyelitis by Early Stretching with Aid of Curare. V. H. Raisman and J. Schneiderman. —p. 93.
*Causes of Failure in Surgical Fenestration: Follow-Up of 3,000 Consecutive Cases. G. E. Shambaugh Jr.—p. 104.
Complications Following Vaginal Hysterectomy. V. P. Mazzola, N. J. Mazzola and F. V. Mitchell.—p. 110.

Early Stretching with Aid of Curare in Poliomyelitis.—Between 1946 and 1948, 136 patients with acute and subacute poliomyelitis were treated by early stretching with the aid of curare on the orthopedic service of the Queens General Hospital in New York. Curare was given intramuscularly every eight hours, usually starting with 0.9 units per kilogram of body weight, with a gradual increase to a maximum of 1.5 units. Three quarters of an hour, one and one-half hours, and two and one-half hours after each daytime injection, the patient was stretched by a physical therapy technician. The greatest effort was exerted in stretching the heel cords, hamstrings, adductors, quadriceps, back, and neck extensors. Results demonstrated that maximum improvement is facilitated by vigorous and continued stretching with curare because normal muscle length is retained; fixed deformities are prevented; no obstacle exists to recovery of power by weak muscles; casts and braces are rendered unnecessary; and circulatory disturbances are not observed. A follow-up survey, two to six years after their discharge from the hospital, of about 75% of the patients revealed that stretching must be continued indefinitely; otherwise tightness or shortening will return, thereby preventing weak muscles from improving and permitting contractures or even deformities to develop. The relation between muscle shortening and unequal leg length is pointed out. There is no evidence that either intensive stretching or early exercise produces any damage to muscles. On the contrary, intensive stretching, facilitated in the early stages by the use of curare plus early exercise, is the best orthopedic method of treatment of acute and subacute poliomyelitis.

Failure in Surgical Fenestration.—In 233 (7.5%) of 3,091 consecutive fenestration operations performed at Northwestern University of Chicago, there was initial failure to achieve a significant hearing improvement of more than 10 db. for the speech frequencies. Postoperative serous labyrinthitis with permanent depression of cochlear function accounted for initial failure in 165 cases, or 70% of all initial failures, and that despite an appreciable reduction in the incidence of this complication with improvements in technique. Incorrect selection of patients for surgical treatment accounted for 62 (27%) initial failures in the 3,091 operations. Considering the fact that successful fenestration leaves a residual conductive loss of approximately 25 db., owing to absence of an impedence-matching lever mechanism, fenestration cannot be expected to result in more than 10 db. of improvement if the preoperative conductive loss, measured by the air-bone gap, is less than 35 db. With better appreciation of the need for an adequate preoperative air-bone gap of 40 to 60 db. and with improved methods of estimating cochlear reserve, the considerable number of failures attributable to incorrect selection should be greatly decreased in the future. Obvious technical errors during the surgical intervention accounted for six failures (0.2%); in one case the endolymphatic labyrinth was torn, with bleeding in the ampullar region, and in five cases the tympanic membrane suffered a large permanent laceration. Up to now 183 (6%) of the 3,091 operations achieved but failed to maintain a hearing improvement on follow-up examinations for longer than 10 years, and were therefore classified as late failures. Although one should not expect fenestration to influence the cochlear nerve degeneration as-

sociated with otosclerosis, experience suggests that it may be somewhat delayed by successful fenestration. Statistically the fenestration operation results in a maintained significant improvement of hearing in about 85% of patients followed for 6 months to 14 years. The final decision as to the value of this improvement to a particular patient depends on the degree of loss of hearing before the operation, the success that the patient might have had in using a hearing aid, and the hearing needs of that patient in his occupation. The two major problems that await solution are further reduction of the incidence of postoperative serous labyrinthitis, and a method for eliminating the 25 db. of residual conductive loss after successful fenestration that still prevents the restoration of entirely normal hearing in patients with normal function of the cochlear nerve.

Journal of Investigative Dermatology, Baltimore

20:1-66 (Jan.) 1953

Allergic Factors in Production of Leukemids. R. W. Goldblum, F. C. Bocobo and A. C. Curtis.—p. 1.
Hemolytic Serologic Reaction Without Complement as Possible Test for Syphilis. H. A. Cohen.—p. 5.
Metal Content of Skin, Nails and Hair. R. W. Goldblum, S. Derby and A. B. Lerner.—p. 13.
Water Soluble Vitamins in Normal Human Skin. T. H. Lee, A. B. Lerner and R. J. Halberg.—p. 19.
Note on Fingernail Growth. W. B. Bean.—p. 27.
Studies of Skin-Hypersensitivity to Lanolin. M. B. Sulzberger, T. Warshaw and F. Herrmann.—p. 33.
Effects of Resin of Euphorbium on Verrucae Plantares—Human and Animal Experimentation. R. W. Goldblum and A. C. Curtis.—p. 45.
Histopathologic Studies of Pigmented Nevi in Children. O. C. Stegmaier Jr. and H. Montgomery.—p. 51.

Journal-Lancet, Minneapolis

73:1-40 (Jan.) 1953

Crime and the Doctor. K. Simpson.—p. 1.
Splenic Cysts. G. D. Icenogle.—p. 6.
Regional Coordination Helps Small Hospitals. C. E. Caven.—p. 9.
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Some Physiological and Clinical Aspects of 24-Hour Periodicity. F. Halberg.—p. 20.

Journal of the Mount Sinai Hospital, New York

19:619-698 (Jan.-Feb.) 1953

Recent Advances in Theory of Mechanism of Blood Coagulation. M. Stefanini.—p. 619.
Experimental Approaches to Psychodynamic Problems. J. H. Masserman.—p. 639.
Extracellular Compartment: Comparison of Chloride and Inulin Spaces. L. B. Turner and M. F. Levitt.—p. 653.
*Perforation of Pyriform Sinus; Sequela of Endotracheal Intubation. M. H. Adelman.—p. 665.
Correlation of Dental Abnormalities in Hypo-Pituitarism. J. A. Salzmann and S. L. Wein.—p. 668.
Myoma of the Second Portion of Duodenum: Case Report. A. A. Bakst.—p. 677.
*Epilepsy and Electroencephalogram. L. Greenstein.—p. 683.

Perforation of Pyriform Sinus in Endotracheal Intubation.—The man, whose history is presented, gave the impression that he had an expanding lesion of the right inferior frontotemporal region and chronic bronchiectasis. On Oct. 18, 1949, he was prepared for a subtemporal decompression. Preanesthetic medication consisted of 0.4 mg. of scopolamine hydrobromide and 250 mg. of amobarbital (amytal®). After the nose, pharynx and larynx were anesthetized with 5% cocaine, and a soft Magill endotracheal tube was inserted blindly into the trachea with some difficulty; an initial attempt was unsuccessful. The intubation was performed in order to have a satisfactory airway if general anesthesia was required. On the fourth postoperative day the patient had dysphagia and expectorated large amounts of gray, mucopurulent material. The dysphagia and productive cough persisted through the remainder of the illness. On Nov. 1, 1949, a craniotomy was performed. A mass in the right temporal lobe was excised. The patient's condition was good for four days. On the fifth postoperative day, he suddenly became stuporous, dyspneic, and cyanotic; coarse bubbling rales were heard in all lung fields. Death occurred one hour after onset of stupor and cyanosis. The postmortem examination revealed in the right paratracheal region, communicating with the posterior portion of the right pyriform sinus by a small perforation, a large, poorly delineated abscess cavity, which contained 75 cc. of foul, grayish pus. The abscess cavity dissected through tissue planes anteriorly to involve the larynx and posteriorly around the tracheal cartilages; it extended superiorly and retropharyngeally almost to the base of the sphenoid bone. Confluent bronchopneumonia of the left lower lobe, perforated peptic ulcers of the duodenum, and arteriosclerotic heart disease were noted. The perforation of the pyriform sinus was an unexpected postmortem observation. A less stuporous patient might have exhibited symptoms of the neck infection. The question whether or not the perforation contributed to the patient's death could not be answered. Trauma to the pyriform sinus, during the initial attempt at blind intubation, was presumably the cause of the perforation. The site of the perforation indicated that the tip of the endotracheal tube lacerated the mucosa of the pyriform sinus. The perforation probably occurred in the early postoperative period rather than at the time of intubation. Anesthesiologists differ in their attitude to blind intubation, but most otolaryngologists condemn it, and some believe that blind intubation is excusable only in cases in which the laryngoscope cannot be used.

Epilepsy and the Electroencephalogram.—The most important clinical application of the electroencephalogram is to confirm or rule out the existence of an epileptic disorder. The fact that normal records do occur in known epileptics is disturbing, but this disturbing feature can be reduced by attention to various clinical and historical features associated with normal and abnormal records. To ascertain some of these features Greenstein studied 100 epileptics. Irrespective of the types of seizures they had, patients with near relatives suffering from epilepsy rarely showed a normal electroencephalogram. In the absence of any familial history of epilepsy, a normal electroencephalogram was frequently found in epileptics, irrespective of the types of seizures, providing the seizures did not include petit mal. The earlier the age of the onset of seizures the greater the likelihood of the presence of an abnormal electroencephalographic record. Anticonvulsants decreased the number of abnormal electroencephalographic records. No correlation was found between the electroencephalogram and (*a*) frequency of seizures, (*b*) age at the time of recording, (*c*) duration of illness, (*d*) time of day of attacks, or (*e*) any combination of type of seizures providing they did not include petit mal.

Maine Medical Association Journal, Portland

44:1-32 (Jan.) 1953

*Erysipeloid—Method of Treatment. P. O. Gregory.—p. 1.
Ophthalmic Surgery in Thyroid Pathology (Dysfunction). E. B. Spaeth.—p. 2.
The Thymus in Early Infancy. G. E. Dash.—p. 7.
Simple Treatment of Simple Fractures. J. P. Goodrich.—p. 9.
Gastro-Intestinal Bleeding—Case Reports. P. O. Gregory.—p. 14.
Cases of Probable Suicide in Young Persons Without Obvious Motivation. A. Warren Stearns.—p. 16.

Treatment of Erysipeloid.—The commonest form of erysipeloid seen in fishermen in Maine usually appears on the hand, occasionally in the form of paronychia. It causes itching and burning, and appears as a smooth, circumscribed, more or less edematous plaque, at times with elevated irregular borders. It slowly extends over the fingers and up to the wrist, usually only on the dorsal surface. Lymphangitis and lymphadenitis occur in some cases. The incubation period is about one day. Fever and malaise and severe, deep-seated pain, accompanied by throbbing, may incapacitate the patient at the onset. Gregory treated in all about 600 cases of this form of erysipeloid in Maine fishermen. In earlier years ointments containing ichthyol or ichthammol were used; later sulfonamides were tried without much success. Recently he has been giving patients with this form of erysipeloid 300,000 units of penicillin intramuscularly on two or three successive days and locally neomycin sulfate (0.5 mg. per gram). To this treatment 90% of the patients respond satisfactorily. Usually the symptoms are gone in not more than three days from the start of treatment.

FOREIGN

Acta Cardiologica, Brussels

7:587-694 (No. 6) 1952

*Commissurotomy and Mitral Stenosis. R. Stas, P. Soulie, M. Servelle and J. Rougeulle.—p. 587.
*Arrhythmias During Catheterization of the Heart in Man. U. Episcopo.—p. 594.
Arterial Hypotension in Course of Intravenous Infusion of Histamine. J. Lecomte.—p. 619.
Effects of Pyridine Derivatives Such as Nicotinic Acid, Nicotinamide, Nikethamide and Beta-Pyridylcarbinol, on Circulation. F. M. Goksel.—p. 630.

Commissurotomy and Mitral Stenosis.—Commissurotomy according to Bailey and co-workers' technique was performed on 38 patients with mitral stenosis. A left lateral thoracotomy was followed by injection of procaine hydrochloride into the pericardium and into the base of the auricular appendage, the passing of a purse-string suture about the appendage through the injected area, and clamping, opening of the tip, mitral exploration, and digital separation of the valve leaflets or actual division with the knife. In four patients the valves were extensively calcified. If there is calcification of the commissures only and one is able to enlarge sufficiently the orifice, the result will be satisfactory. It will be less satisfactory if calcification extends to the insertion of the valves. Preoperative studies of the circulatory dynamics are indispensable. Catherization of the cavities of the right heart associated with quantitative determination of the respiratory exchanges and measurement of the mitral surface gave valuable information, which was confirmed at surgical intervention. Commissurotomy should be performed only on young patients with sinus rhythm, dyspnea during exercise, pulmonary edema, pulmonary hypertension, and a mitral valve that has only a small surface. Patients with recent attacks of rheumatic fever, infectious endocarditis, heart failure, and arteritis of the pulmonary artery should not be operated on. Of the 38 patients, 2 died, one within 10 hours and the other 3 days after the surgical intervention. In the first patient the heart and the liver were enlarged, there was cyanosis, and the maximum pressure in the right ventricle and the pulmonary artery was 90 mm. Hg. Pulmonary arteritis associated with mtiral stenosis is a contraindication to commissurotomy. In the second patient death resulted from acute edema. Results obtained in the remaining 36 patients were satisfactory; late functional results were particularly good since patients were able to work 16 months after the operation. Contrary to the observations reported by other workers, diminution of pressure in the cavities of the right heart occurred only six to nine months after the surgical intervention.

Arrhythmias During Cardiac Catherization.—A series of 40 catheterizations of the heart were done under electrocardiographic observation in 33 patients between the ages of 4 and 48; 1 patient was catheterized three times, and 5 were catheterized two times. Of the 33 patients, 17 had congenital heart disease, and 16 had rheumatic mitral stenosis, which in 7 was associated with aortic regurgitation. Except for three rheumatic patients with auricular fibrillation, all the other patients had sinus rhythm prior to cardiac catheterization. Various disturbances of cardiac rhythm occurred during 38 of the 40 cardiac catheterizations. The over-all percentage of arrhythmias was 95%. In one patient, a 9-year-old girl with congenital heart disease, a disturbance of cardiac rhythm occurred during the extracardiac passage of the catheter in the left median basilic vein to the axillary vein. A definite relationship between the location of the catheter tip and the type of the catheter-induced arrhythmia was demonstrated in the other patients. Particularly critical stages of the procedure were passage of the catheter through the tricuspid valve, arrest of the tip of the catheter against the interventricular septum and the ventricular walls, and penetration into the pulmonary artery. Severe arrhythmias observed included one case of supraventricular tachycardia in a patient with congenital heart disease and Wolff-Parkinson-White syndrome, one case of auricular fibrillation which persisted thereafter, one transient complete auriculoventricular block, and one transient right bundle branch block. The origin of the observed arrhythmias is discussed briefly. As a result of his observations the author states that cardiac catheterization should always be done under constant electrocardiographic control, in order to recognize the onset and nature of the arrhythmias that may develop, and to institute proper therapy without delay.

Acta Medica Orientalia, Jerusalem

11:213-248 (Nov.-Dec.) 1952

Mumps-Meningoencephalitis: Observations from General Hospital. K. Marberg.—p. 213.
Mumps-Meningoencephalitis in Children. W. Falk.—p. 220.
*Systemic Mumps. E. Israel.—p. 231.
Laboratory Procedures in Mumps Infection. H. Bernkopf.—p. 241.
Epidemiology of Mumps in Israel. G. Kallner.—p. 244.

Systemic Mumps.—In most virus diseases the virus involves all systems of the body from the onset. As a result of his experiences and of an extensive review of the literature on mumps, the author assumes that the mumps virus is no exception. On clinical grounds there is no doubt that mumps is a general infection, the virus of which can attack every organ and has a special predilection for the salivary glands, the generative organs, the pancreas, and the nervous system. So-called complications are, therefore, acute manifestations of the disease that in most cases appear after swelling of the parotid gland. There are however many cases in which involvement of other organs precedes parotitis, occur at the same time, or make their appearance without any swelling of the parotid gland. A fatal case of myelitis of Landry's type after mumps in a 37-year-old woman and a case of diabetes after mumps in a 16-year-old girl are reported in detail. In children involvement of the testes is rare, but in one of every three adult patients the testes are involved. In some of the patients the testis becomes enlarged to the size of an orange, is painful but soft, and has the appearance of a big hydrocele, while in the majority of patients the testis is stony hard, more painful, but seldom reaches the size of the former. In almost every one of the author's cases of "hard orchitis" some kind of atrophy occurred, while in the soft, hydrocele-like orchitis such consequences were very rare. During a widespread epidemic of mumps in Israel in 1951 serial electrocardiograms were made in 20 children between the ages of 6 and 10 with mumps; none of them had signs and symptoms of cardiac involvement, but changes in the electrographic recordings such as prolonged PR interval, inverted QRS complex and negative T waves were observed, providing additional proof that mumps is a systemic disease capable of affecting the heart. The use of the term epidemic parotitis instead of mumps is therefore misleading. It narrows the conception of this disease and should be abandoned. There is ample evidence from laboratory studies that supports the author's concept of mumps as a systemic disease. Mumps virus has been isolated from the blood stream, the cerebrospinal fluid, and from the testis, and antibodies have been observed in the hydrocele fluid. The fact that it almost always gives a life-long immunity shows that in this respect, too, mumps follows the same pattern as other systemic diseases. There are, however, patients who after an attack of mumps seem to suffer from a reduced resistance to the mumps virus; an illustrative case in a 48-year-old man who had three recurrences of mumps and orchitis within three years after the first attack is reported.

Archivio Italiano di Chirurgia, Bologna

75:411-496 (No. 6) 1952. **Partial Index**

Experiments on Reconstruction of Bronchial Tree with Homografts and Autografts. A. Bonamone and F. Natellis.—p. 411.
Aneurysm of Splenic Artery. S. Milone.—p. 427.
Lumbar Hernia: Clinical Contribution and Pathogenetic, Anatomic, and Therapeutic Considerations. G. Bocchetti.—p. 441.
Cyanosis Caused by Arteriovenous Aneurysm of Lung. R. Paolucci di Valmaggiore and G. Folanini.—p. 455.
*Use of Placenta To Repair Loss of Bronchial Tree Substance: Observations and Experimental Studies. F. Natellis.—p. 463.

Placenta in Reconstruction of Bronchial Tree.—Grafts of fresh placenta washed in isotonic sodium chloride solution and kept at a temperature of from 2 to 4 C were used by the author as plastic material in experiments to rebuild the bronchial tree in which injuries had been deliberately produced. The operation was performed in 10 dogs under intratracheal anesthesia; in

six of them the placenta was applied over a wide gap produced in the primary bronchus, and in four over the bronchial stump resulting from removal of a lobe. In the dogs of the first group after the lung was collapsed, a 1.5 cm. by 0.5 cm. fenestration was made in the main bronchus and the placenta was placed over it and fastened with a few fine silk sutures. Sulfonamides and penicillin were sprayed over the operative field before the external sutures were applied. In the dogs of the second group the left upper lobe was removed and the placenta was placed over the stump like a hood and was fastened with silk sutures. The operation was successful in three dogs whereas one died seven days later because of empyema. There were no postoperative complications in the remaining dogs, and all the animals were in good general condition until the day they were killed (4 to 90 days later). Four days after the operation there was no microscopic evidence of fusion of the chorion tissue with the surrounding bronchial tissue. Twelve days postoperatively the surrounding tissue showed great proliferation of young connective tissue elements, and some chorionic villi were still visible. After three months cicatrization and epithelization had already occurred on the operated area and the bronchial epithelium had covered the placenta that was no longer visible. The placenta tissue, having served as a tampon for the bronchial fenestration, had undergone lysis with a subacute inflammatory reaction. Around the 45th day a centripetal epithelization of the plastic area began which was completed within two months and in the third month the epithelium of the respiratory tract was again normal. In the meantime, however, the presence of the placenta in the operated area had prevented the severe complications secondary to injuries to the respiratory tract. It is hoped that operations with the use of placenta grafts in similar instances may be possible in man.

British Journal of Experimental Pathology, London

33:513-624 (Dec.) 1952. Partial Index

Gastric Secretion of Blood Group Antigen in Relation to Acid Secretion. D. A. Osborn.—p. 513.

Protein and Nucleic Acid Contents of Rat Livers one Day After Single Injection of Carbon Tetrachloride. R. M. Campbell and H. W. Kosterlitz.—p. 518.

Trypan Blue-Induced Tumours of Rats. C. L. Simpson.—p. 524.

Two Heat-Labile Factors in Normal Sera Which Neutralize Variola Virus. K. McCarthy and W. D. Germer.—p. 529.

Haemagglutination with Virus of Murray Valley Encephalitis (MVE). F. Macdonald.—p. 537.

Effect of Certain Non-Specific Factors on Production of Antitoxin. M. Barr and A. T. Glenny.—p. 543.

Influence of Cortisone on Regeneration of Tendon and Tracheal Epithelium in Rat. R. C. Buck and D. L. Wilhelm.—p. 562.

Comparison Between Intranasal Instillation and Natural Inhalation as Means of Infecting Mice with Influenza Virus. R. H. Bowers, O. L. Davies and E. W. Hurst.—p. 601.

Study of Chick Embryo Lesions Produced by Influenza Viruses. J. C. N. Westwood.—p. 610.

British Medical Journal, London

1:117-172 (Jan. 17) 1953

Serous Hepatosis: Pathogenesis of Hepatic Fibrosis in Jamaican Children: Preliminary Report. K. R. Hill, K. Rhodes, J. L. Stafford and R. Auh.—p. 117.

*Treatment and Prognosis of Acute Perforated Peptic Ulcer. F. A. Jones and R. Doll.—p. 122.

Granuloma of Nose and Periarteritis Nodosa. H. J. M. Stratton, T. M. L. Price and M. O. Skelton.—p. 127.

Segmental Aspiration Pneumonia and Bronchiectasis. K. D. F. Morle and P. W. Robertson.—p. 130.

*Influence of Oestradiol Benzoate and Orchidectomy on Reticulo-Endothelial System. T. Nicol and A. Abou-Zikry.—p. 133.

Response in Dogs to Relaxants Derived from Succinic Acid and Choline. L. W. Hall, H. Lehmann and E. Silk.—p. 134.

Effect of Pseudo-Cholinesterase Level on Action of Succinylcholine in Man. F. T. Evans, P. W. S. Gray, H. Lehmann and E. Silk.—p. 136.

Breast-Feeding in Oxford Child Health Survey: Part I—Study of Maternal Factors. C. Westropp.—p. 138.

Acute Perforated Peptic Ulcer.—At the gastroenterological department of the Central Middlesex Hospital 715 patients were treated for acute perforated peptic ulcer between 1938 and 1951. While up to 1942 the operative mortality was commonly 20%, there has been a striking fall in the mortality rate during the following years. Since 1947 there does not seem to have been any appreciable change in the mortality rate. Of the 715 patients, 340 were treated between 1947 and 1951 and among them, including those undiagnosed till after death, the mortality rate has been 7.9%. Of the 340 patients 20 were not operated on, mostly because the condition was not diagnosed or the patient was too ill; 17 died. The operative mortality in the remaining 320 patients was 3.1%. Nineteen patients were treated by immediate partial gastrectomy without a death; in the first six months of 1952 the number of patients so treated has been increased to 30. The other 301 were treated by simple surgical closure of the perforation with 10 deaths, a mortality rate of 3.3%. The concurrent association of hemorrhage and perforation was successfully treated by both simple closure and by immediate partial gastrectomy. Seventy-four patients treated by simple closure for perforated gastric ulcers and an equal number of comparable patients with perforated duodenal ulcers were followed up after 2 to 12 years. Of those with gastric ulcer, 6 could not be traced, 17 were dead, and 51 were living; of those with duodenal ulcer, 4 could not be traced, 12 were dead, and 58 were living. Eight (11%) of the patients with gastric ulcer, but none of those with duodenal ulcer, died of carcinoma of the stomach. Seven (5%) of the 138 patients with gastric or duodenal ulcer who could be traced died of peptic ulcer. The late prognosis among the known survivors was unaffected by the sex or age of the patients or the site of the ulcer. It was affected by the length of history prior to the perforation and by previous diagnosis of an ulcer; 41% of those with a history of dyspepsia of more than a year and 50% of those with a previous diagnosis of an ulcer were reoperated on during the follow-up period. The advantages and disadvantages of the three current methods of treatment, namely, medical management with gastric aspiration, simple surgical closure, and immediate partial gastrectomy, are discussed, and it is concluded that no one method should be the method of choice in all cases. In any one series all three methods have their proper place. Tentative conclusions about the indications for choice of method in individual cases are suggested.

Effect of Estradiol Benzoate and Orchidectomy on Reticuloendothelial System.—Twenty-two intact male guinea pigs and 14 guinea pigs with bilateral orchidectomy were divided into groups in which each animal received one daily intramuscular injection of 0.5 mg. of estradiol benzoate for one, two, three or four weeks, and one daily subcutaneous injection of trypan blue during the last six days of the hormone treatment, and were killed the following day. As controls 6 additional intact animals and 10 castrated animals were given one daily subcutaneous injection of trypan blue for six days and were killed the next day. The activity of the reticuloendothelial system of the animals was measured by the number of dye-bearing cells in the spleen, liver, lymph nodes and prostate, and the intensity of the vital staining in the organs and tissues was examined. Results showed that in both the intact and the animals in whom orchidectomy had been done estradiol benzoate produced hyperplasia and stimulation of the reticuloendothelial macrophages, mainly in the spleen, liver, lymph nodes, and prostate and that the degree of stimulation was roughly proportionate to the duration of the hormone treatment. In the animals whose testes had been removed the reticuloendothelial system was more active, and estradiol benzoate produced a greater response than in the intact animals. In the prostate, the thin layer of fibrous tissue that normally surrounds the prostatic acini became progressively thicker with hormone treatment, and the prostate showed the greatest degree of fibrosis in the castrated animals that received estradiol benzoate for four weeks. These results, therefore, show that estrogens and orchidectomy both stimulate the reticuloendothelial systems and in this manner probably increase the general resistance against cancer; that in the prostate they induce the appearance of macrophages and later of fibrous tissue, which acts as a local defense against the spread of cancer; and that estrogenic treatment combined with orchidectomy produces a more intense result than either alone.

Canadian Medical Association Journal, Montreal

68:1-96 (Jan.) 1953

Place of Psychiatrist in Community Medical Services. R. O. Jones.—p. 1.
Present Status of Anticoagulants in Thrombo-Embolic Disease. E. S. Mills.—p. 6.
Tonsil and Adenoid Problem. G. E. Tremble.—p. 9.
*Use of ACTH, Cortisone and Salicylates in Treatment of Acute Rheumatic Fever. R. D. Rowe, A. D. McKelvey and J. D. Keith.—p. 15.
Some Problems of Method in Neurology. F. M. R. Walshe.—p. 21.
The Aged Mental Hospital Patient. W. Forster.—p. 29.
Clinical Orientation to Alcoholism. R. G. Bell.—p. 33.
Progress in Radiotherapy. I. H. Smith.—p. 40.
Simplified Technique for Nailing Fractured Neck of Femur. C. McPherson and G. W. Kruger.—p. 43.
Appendicitis. C. Gardner.—p. 46.
Cortisone in Treatment of Acute Self-Limited Dermatoses. N. M. Wrong and R. C. Smith.—p. 50.
Prevention of Tuberculosis Among Nurses. R. G. Ferguson.—p. 56.

Corticotropin, Cortisone, and Salicylates in Rheumatic Fever.—A controlled therapeutic trial was carried out on 41 patients with rheumatic fever ranging in age from 4 to 41 years. They were treated with one of three agents by random allocation. Corticotropin (ACTH) was given in total doses of 2.6 gm. over a period of six weeks by intramuscular injection every 6 hours. The daily doses were gradually reduced from 120 mg. to 5 mg. Acetylsalicylic acid (aspirin) was given in doses of 1 grain (65 mg.) per pound (0.453 kg.) of body weight for the first two days, ⅔ grains (45 mg.) per pound for five days; then ½ grain (32 mg.) per pound for the next five weeks. Cortisone was given by intramuscular injection in total doses of 4.1 gm. over a period of six weeks, the daily dose being gradually reduced from 300 mg. on the first day to 50 mg. on the last 7 days. Certain beneficial effects were encountered in all three groups. The temperature and arthritis are usually controlled by all three preparations. The sedimentation rate comes down more rapidly with the hormones than with the salicylates, but this in itself does not indicate any benefit to the patient. When the hormones were first tried it was hoped that there would be alleviation of the damage to the heart muscle and valves. This study has not shown any significant difference between hormones and salicylates as far as one can measure the effects on the heart. The most noticeable difference between the three treatment groups was the response to cessation of therapy. The hormone group had a rebound of the sedimentation rate in over two-thirds of cases. One-third had associated fever and one-sixth arthritis. One patient died during this rebound period. No fever or arthritis occurred in the salicylate group and slightly over one-third had rebounds of the sedimentation rate. The authors feel that the salicylates are the more practical agent in rheumatic fever.

Deutsche medizinische Wochenschrift, Stuttgart

78:85-116 (Jan. 16) 1953. Partial Index

Pathologic Aspects of Parathyroids and of Calcium and Phosphate Metabolism. G. Fanconi.—p. 85.
Cerebral Lesions After Dystrophy and Exhaustion. F. W. Bronisch.—p. 89.
Clinical Aspect and Therapy of Diffuse Cranial Hyperostosis. K. Schatter.—p. 90.
*Clinical Experiences with Triethylene Melamine. W. Pribilla.—p. 95.

Clinical Experiences with Triethylene Melamine.—Pribilla discusses the chemistry and pharmacology of triethylene melamine and briefly reviews the first clinical experiments. The drug was at first given intravenously, and was better tolerated than the nitrogen mustards. More recently intravenous administration has been largely abandoned in favor of the simpler oral administration. Triethylene melamine should be given with water about one hour before breakfast. Although the drug is generally well tolerated, nausea and vertigo are occasionally observed some hours after ingestion. The dosage requires individualization; to patients with fair general condition the author usually gives 2.5 mg. on the first and 5 mg. on the second day, and then he waits 8 days to see what effect this dose has on the blood picture. Excessive dosage may cause panmyelopathy. The author has used the drug so far in 8 patients with lymphogranulomatosis (Hodgkin's disease), in one with lymphosarcoma, in one with chronic lymphoid, and in one with chronic myeloid leukemia. The histories of some of these patients are described. Of the eight patients with Hodgkin's disease, three showed temporary improvement and the patient with lymphosarcoma and the one with lymphoid leukemia also showed improvement. Treatment with triethylene melamine is better tolerated by and is more agreeable to patients than is treatment with nitrogen mustard or roentgen irradiation.

Gynaecologia, Basel

134:361-420 (Dec.) 1952. Partial Index

*Atrophy of Uterine Mucosa and Ozena Caused by Dysfunction in Hypophyseo-Hypothalamic Region: Favorable Results of Treatment with Vitamin E and Gonadotropin. E. Klees and H. G. Müller.—p. 361.
*Value of Vitamin E in Treatment of Toxaemia of Late Pregnancy. J. L. Mastboom and A. Sikkel.—p. 391.
Effect of Anemia on Duration of Labor. H. Zilliacus and T. Putkinen.—p. 397.

Atrophy of Uterine Mucosa and Ozena Caused by Dysfunction in Hypophyseohypothalamic Region.—Secondary amenorrhea can be caused by various disturbances in the genital sphere as well as by psychic and neurosympathetic disorders and by dysfunction in the hypophyseohypothalamic region. On the basis of the histories of eight cases, Klees and Müller discuss genital dysfunction of central origin and the interrelation of function and dysfunction of the genitalia and that of other organs. In the cases reported, the centrally induced genital disturbances were accompanied by atrophy of the nasal mucosa and the authors direct attention to the ontogenetic relations between the nasal mucosa and the hypophysis, which seems to explain this association of dysfunction of the nasal mucosa and a hypophyseohypothalamically induced disorder of the genital cycle. The treatment of these centrally induced functional disturbances should, as is demonstrated in some of the case histories presented, never consist of an ovarian substitution therapy in the form of ovarian hormones, because the prolonged use of such treatment will lead to the inhibition of the production of gonadotropic hormones by the hypophysis, and beyond that, possibly, to the impairment of the production of other hypophyseal hormones. Since it has been demonstrated that vitamin E has a stimulating effect on the hypophyseohypothalamic system, the authors employed vitamin E together with gonadotropin in the treatment of the patients in whom amenorrhea was associated with dysfunction of the nasal mucosa in the form of ozena. Vitamin E was given in daily doses up to 40 mg. for a number of weeks until a total of 6 gm. had been given. Gonadotropin was usually given twice weekly in individual doses of from 1,000 to 3,000 units. After an interval of two weeks, the course of treatment may be repeated if necessary.

Toxemia of Pregnancy not Influenced by Vitamin E.—Several reports on favorable results obtained with vitamin E in the toxemias of late pregnancy and its value as a prophylactic against premature detachment of the placenta induced Mastboom and Sikkel to study the use of vitamin E for these purposes. A survey of the literature did not reveal any proof that vitamin E has a beneficial effect on toxemia of late pregnancy. Its value in the prevention of premature detachment of the placenta or a relation between a low alpha tocopherol level in the serum and toxemia are not demonstrated either. Administration of vitamin E (300 mg. daily) to seven pregnant women with preexistent vascular-renal disease and to eight with a superimposed toxemia produced no change. The blood pressure, edema, and albuminuria showed no improvement other than could be expected from bed-rest and a salt-free diet alone.

Journal of Laryngology and Otology, London

67:1-58 (Jan.) 1953

Respiratory Air Currents and Their Clinical Aspects. A. W. Proetz.—p. 1.
Interstitial Irradiation of the Larynx: Report of Eight Cases. M. Mason and F. C. Ormerod.—p. 28.
Cholesteatoma: Problem of Operation. R. G. Hughes and W. McKenzie.—p. 38.
Method of Dilatation of Strictures of the Oesophagus. R. M. Harvey and V. E. Negus.—p. 43.

BOOK REVIEWS

Man: The Chemical Machine. By Ernest Borek. Cloth. $3. Pp. 219, with illustrations. Columbia University Press, 2960 Broadway, New York 27, 1952.

The author brings the reader up-to-date on recent developments in physiological chemistry. The first chapter shows that the chemical elements in the living organism are the same as those in the dead; the essential differences in chemistry of living and dead matter are gradually brought out in the nine following chapters. These deal with enzymes, vitamins, sugars, isotopes, amino acids, blood corpuscles, immunity, genes, and the activity of nerve cells. The concluding chapter summarizes the rewards that have accrued from the study of biochemical mechanisms and outlines the possibilities of even greater benefits from future research. Use of chemical formulas has been avoided so as not to alienate readers who are not interested in such technicalities. In other respects, however, the writing is concrete and specific; the author provides names, dates, places, and an index. The book will undoubtedly bring inspiration and pleasure to many general readers. It can also be read profitably by physicians seeking an orientation in recent developments of biochemistry.

Disorders of the Circulatory System. Edited by Robert L. Craig, M.D. Symposium presented at twenty-fourth graduate fortnight of New York Academy of Medicine, October eighth to nineteenth, 1951. Cloth. $5.50. Pp. 305, with 87 illustrations. The Macmillan Company, 60 Fifth Ave., New York 11, 1952.

The annual graduate fortnight of the New York Academy of Medicine has become an important medical event. For the past two years the papers presented at the meeting have been made available in book form. Contributions to this volume cover many of the outstanding clinical and laboratory investigations in the field of cardiovascular diseases. Topics of the papers are of current interest and include recent advances in pathogenesis of arteriosclerosis, surgical treatment of cardiac defects, biochemistry of heart muscle, the role of connective tissue in diseases of the cardiovascular system, the mechanism of congestive heart failure, new electrocardiographic data, factors associated with hypertension, and, finally, a thorough review of circulatory responses to life situations. Some of the published lectures are illustrated, and many have references for those who wish to refer to the original articles. This is a book that should be in the library of every physician who is interested in the latest advances in the field of cardiovascular diseases.

La diagnostica funzionale delle epatopatie. Di A. Gambigliani Zoccoli e C. Franzini. Paper. 2000 lire. Pp. 303, with 131 illustrations. Edizioni Minerva medica S.A., Turin, 1952.

The author presents first the basic physiopathology of biliary pigment and then pathogenesis and classification of jaundice. The discussion is well documented with brief presentation of many clinical cases, and these are related to liver morphology (biopsy) and to functional disturbance. The most interesting contribution is the chapter on the dual (mesenchymal and the parenchymal) aspects of hepatic damage. Mesenchymal damage is represented as actually being a histiocytic hyperplasia, leading to annular fibrosis, without evidence of inflammatory process, but with an outstanding hyperglobulinemia. In these forms initially the parenchymal function is normal and the albumin values are either normal or only slightly decreased. In discussing the parenchymal insufficiency the author questions the value of flocculation tests as specific indicators of primary hepatic lesions, since the results obtained may be indicative of other conditions. Once it has been determined that the basic disease is hepatic, however, flocculation tests are valuable. The relationship of flocculation tests to clinical manifestations and liver biopsy is well illustrated. Technical discussions include the technique of liver biopsy. The book is clearly written, well illustrated, and has an extensive bibliography. It represents a valuable contribution interesting to the clinician and the clinical pathologist.

Operating Room Technic, St. Mary's Hospital, Rochester, Minnesota. Fourth edition. Cloth. $6.50. Pp. 345, with 219 illustrations. W. B. Saunders Company, 218 W. Washington Sq., Philadelphia 5; 7 Grape St., Shaftesbury Ave., London, W.C.2, 1952.

The first edition of this small book appeared in 1924; the present edition has been completely rewritten. Additions in sections on peroral endoscopy and urology are timely. Welcome features are diagrams indicating the positions of each member of the surgical team during various operative procedures and illustrations of the positioning of patients. The book is well illustrated, and, although some of the figures are old, they are still accurate. It is unfortunate that the lettering on some of the figures has been reduced so much that the captions are difficult to decipher; an example of this is figure 14 on page 129, dealing with splenectomy. The text has been condensed to the bare essentials of each procedure, and, despite the fact that the material is presented in an extremely abbreviated form, the presentation is good. The book can be recommended as a quick reference for anyone interested in operating room techniques.

Protective Body Mechanics in Daily Life and in Nursing: A Manual for Nurses and Their Co-Workers. By Margaret Campbell Winters, R.N., P.T., Instructor in Nursing, Vanderbilt University School of Nursing, Nashville, Tenn. Paper, loose-leaf, $3.50. Pp. 150, with 393 illustrations by Harold Black and Clifford Johnston. W. B. Saunders Company, 218 W. Washington Sq., Philadelphia 5; 7 Grape St., Shaftesbury Ave., London, W.C.2, 1952.

The nurse, either student or graduate, the physical therapist, and the physician should find this monograph a ready source of practical information. This book is ring bound and is profusely illustrated with line drawings. The first section is devoted to a simple presentation of anatomy and physiology as related to muscular activity. The second part, entitled "Factors Which Influence Body Mechanics," briefly and rather superficially presents topics such as body mechanics of infants, children, and adolescents. The third section, entitled "Application of Principles of Body Mechanics to Nursing," teaches methods of minimizing strain on the musculoskeletal system by using proper methods of lifting, moving, and turning the patient. It also will serve as a guide to the nurse in teaching the patient to effectively and properly help himself.

Hernia: Anatomía, etiología, síntomas, diagnóstico, diagnóstico diferencial, pronóstico y tratamiento. Por Leigh F. Watson, M.D., F.I.C.S. Traducción de la "nueva" 3ª edición en inglés, puesta al día por el autor, con agregados de los traductores doctores Diego E. Zavaleta y Julio V. Uriburu. First Spanish edition. Cloth. Pp. 922, with 472 illustrations. Editorial artécnica, Venezuela 1702, Buenos Aires, 1952.

The Spanish translation of the third English edition of Watson's monumental work on hernia has been accomplished in an admirable manner by two prominent surgeons in Buenos Aires. They have supplemented the original text with descriptions of new methods that have been developed or become popular since the publication of the last English edition, for example, the use of tantalum mesh, bovine fascia, or full-thickness skin, and the Cooper's ligament hernia repair.

The style is smooth and pleasant, the illustrations are clear, and the paper and binding are of a good quality. The translators as well as the publisher deserve the highest praise for their achievement. They have rendered a great service to their Spanish-speaking confrères by making available to them a standard work that has received universal recognition as an authoritative textbook on hernia.

The reviews here published have been prepared by competent authorities and do not represent the opinions of any official bodies unless specifically stated.

QUERIES AND MINOR NOTES

THE VITAMIN HABIT

To the Editor:—*A patient has been taking multivitamin tablets and mineral tablets for three years. Owing to financial reverses she has been unable to buy either preparation. She claims that, since giving up these tablets, she has intermittently experienced feelings of dizziness, numbness, and ringing in the ears. Is it possible that she has built up a need for these preparations and that her future well-being depends on their continued use?* *William K. Dykes, M.D., Los Angeles.*

This inquiry was referred to two consultants, whose respective replies follow.—Ed.

Answer.—Our interpretation would be that the patient has not developed a dependence for vitamin and mineral tablets because she had been taking such preparations. It would be the other way around. Her tissues, like other living things, must have certain chemical substances in order to function properly. Dizziness, numbness, and ringing in the ears could be symptoms of a vitamin deficiency, although they are unusual ones.

Answer.—Why has this patient taken multivitamin tablets and minerals for a long period? Why, after relief from any possible deficiency syndrome, was she not given a diet that would supply all needed substances? These questions carry a lesson; vitamins and minerals in pure form are sometimes needed, acutely needed, when illness due to a deficiency of these substances actually exists, but, merely for the maintenance of robust health, the person should get his vitamins and minerals from his food. The symptoms described do not indicate that this patient has built up an abnormal need for vitamins and minerals. Instead, the patient may regard her daily vitamin pill as a ritual that prevents her symptoms of dizziness and the like, symptoms that are almost certainly of functional origin. Probably what she needs is reassurance and good food, perhaps with a little phenobarbital.

EFFECT OF SALICYLATES ON HEARING

To the Editor:—*Do salicylates have any lasting effect on the hearing mechanism? If so, does this mean it would be unwise to give adequate doses of salicylates to a patient with acute rheumatic fever who has had some hearing loss as the result of a previous middle ear infection?* *M.D., Utah.*

Answer.—Impaired hearing due to large doses of salicylates or quinine is preceded by ringing in the ears. The dosage that will cause ringing or impaired hearing varies with individual patients. A safe rule to follow is to give salicylates as long as ringing in the ears does not occur. Slight degrees of auditory nerve damage from salicylates and quinine are reversible, so that prompt withdrawal of the drug, should ringing occur, should result in a return of the hearing to the previous level.

PLASMA CELL MASTITIS

To the Editor:—*Have plasma cell tumors of the breast ever been known to become malignant? What is the proper treatment?* *Ellis V. Browning, M.D., Springerville, Ariz.*

Answer.—Plasma cell mastitis is not a neoplastic disease and is, therefore, never malignant; however, the condition is often mistaken for malignant disease, because the mass is typically indurated, with skin attachment and redness, and enlarged axillary nodes may be present. Local excision of the indurated area is the only treatment necessary.

The answers here published have been prepared by competent authorities. They do not, however, represent the opinions of any official bodies unless specifically so stated in the reply. Anonymous communications and queries on postal cards cannot be answered. Every letter must contain the writer's name and address, but these will be omitted on request.

MULTIPLE CHALAZIONS

To the Editor:—*I would like information concerning the prevention and treatment of chalazions. A woman was entirely free of these lesions until two years ago, when they appeared after the birth of her first child. When she became pregnant again, no new lesions occurred; but new lesions again are arising postpartum. Two ophthalmologists advised local application of oxytetracycline ("terramycin") ointment and keeping the meibomian glands unplugged whenever they appear to become infected; however, this has been ineffective. She has had several removed, and, if this is continued, her lids may be badly scarred.*

William G. Osoba, M.D., Stockdale, Texas.

Answer.—Nonsurgical prophylaxis against multiple chalazions consists of massaging the lids in an attempt to expel the contents of the meibomian glands through their acini on the lid margin. A glass rod pressed against the lid on the conjunctival side at the lower border of the tarsal plate and rolled toward the lid margin, with counter pressure applied by the finger on the skin side of the lid, is the most effective method. The local application of astringents, bacteriostatic agents, antibiotics, and irradiation is used judiciously to keep the lid margins and conjunctiva free from pathogenic organisms. It is well understood that they are not effective in preventing the occurrence of a chalazion nor of altering the course of a chalazion once it has appeared. The chalazion is a granuloma, but infection by pathogenic bacteria is unusual, and, consequently, it is not likely that local use of antibacterial agents would be effective in treatment. There may be several causes of chalazion resulting in closure of the acini of the meibomian gland and a foreign body type of reaction in the retained secretion to the retained epithelial cells of the duct. The final stage is liquefaction or a granulation of the gland and its contents and a local thickening of the tarsus. Surgical destruction of the chalazion by incision and drainage followed by application of 2% silver nitrate on a cottonwound applicator is usually sufficient; in case of multiple cysts, an adequate portion of the diseased tarsus should be removed; otherwise recurrences may necessitate further operative interference. With multiple chalazions no hesitation should be felt in doing a widespread careful dissection; fibrous tissue heals gaps in the tarsal plate without deformity.

HEPARIN AND THROMBOSIS

To the Editor:—*I have been confronted with questions about proper dosage of heparin in the prophylaxis and treatment of thrombotic processes. It appears to be a general belief that sufficient heparin should be given to cause obvious prolongation of the coagulation time. Is it necessary to give enough heparin to increase the coagulation time to 30 to 60 minutes?*

Herbert H. Kersten, M.D., Fort Dodge, Iowa.

Answer.—When heparin is injected intravenously in doses of about 50 mg., the highest prolongation of the clotting time will occur about 10 minutes after injection, and the clotting time will return to normal about six hours later. It was formerly believed that the clotting time should always be kept at a high level, and heparin was administered by the intravenous drip method. It has been shown, however, by extensive work in Sweden and in this country, that when intermittent heparin treatment is given, the therapeutic results are the same as those obtained by the constant drip method. At present, it is customary to give 50 mg. of heparin every six hours. At the end of six hours, the clotting time will have returned to normal. A review of the various therapeutic methods may be found in an article by J. E. Jorpes (*Ann. Int. Med.* 27:361, 1947).

EPIDERMOLYSIS BULLOSA

To the Editor:—*A white woman, aged 27, gave birth to a normal boy seven years ago. Four years ago, she gave birth to another boy who, at birth, had unusually long fingernails with inflammation in that region. Pressure bullae developed in various parts of the body, and a diagnosis of epidermolysis bullosa was made. The condition became gradually worse, and the baby died in a few months. Two years ago, the woman gave birth to a girl who had the same long nails and the same condition of epidermolysis bullosa. This baby died after a few months. One year ago, the woman had a miscarriage of undetermined cause. She is pregnant again, with confinement expected in May, 1953. There is no history of this skin condition or similar conditions in the family. The eldest child appears entirely normal. The mother and father are normal and healthy, except that the mother is inclined to obesity, her usual weight being about 175 lb. (79.4 kg.). Her blood has been reported as type A, RH positive. Her pregnancies and deliveries, with the exception of the miscarriage, have been normal. She is taking small doses of thyroid daily and calcium tablets three times a day. What can I tell her about the prospects of the next baby? Is there anything that can be done prenatally to assist her in having a normal child? Is there any treatment for epidermolysis bullosa other than symptomatic treatment?*

M.D., New York.

Answer.—Epidermolysis bullosa is definitely hereditary. Since the patient has already given birth to two babies with the same condition, there is a 50% chance that the same defect will appear in the baby that is expected in May, 1953. Some obstetricians and dermatologists consider it advisable to perform an abortion early in pregnancy in a case similar to this; however, since the baby is to be born shortly there is nothing to do but hope that the baby will be normal. There is no known prophylactic treatment that can be given to the mother. Likewise, unfortunately, there is no satisfactory therapy after a child is born with this condition.

THE UMBILICAL CORD

To the Editor:—*Do the umbilical blood vessels have vasoconstrictor innervation? Is it plausible that the gentlest manipulation of the prolapsed cord in case of a breech presentation, when the head is still high and compression of the cord by the head is unlikely, would provoke vasoconstriction in its blood vessels and thus initiate the breathing reflex in the baby? Is it plausible that merely touching the nonprolapsed cord when doing a vaginal examination in case of a breech presentation would provoke such reflexes?*

George Geoffrey, M.D., Syracuse, N. Y.

Answer.—The umbilical cord proper has no demonstrable nerves. Spivak has demonstrated nonmedullated nerve fibers in the abdominal portion of the umbilical arteries. The umbilical vein has a mechanism for regulating circulation through it known as the folds of Hoboken. Gentle manipulation of the cord should not interfere with the circulation through it. The cord has been palpated at vaginal examination innumerable times without disturbing the circulation through it. Pressure on the cord to interfere enough with blood flow through its vessels to induce sufficient fetal anoxia would be necessary to alter normal fetal respiration.

GASTRIC ULCER

To the Editor:—*I recently had a patient in whom a gastric ulcer developed in five months. What is the shortest time in which a gastric ulcer has developed?*

B. A. Lucking, M.D., Helena, Mont.

Answer.—Gastric ulcers can develop within a matter of minutes to an hour. An acute phase of erosion of the gastric mucosa frequently metamorphoses into a true gastric ulcer. Development over five months, if demonstrable roentgenographically, would lead one to conclude that a subacute or chronic gastric ulcer is the most likely lesion.

GLANDULAR STATUS OF PREGNANT WOMAN

To the Editor:—*What hormone can be given to a nonpregnant woman that will give her the same glandular status as that of a pregnant woman?*

R. S. Lander, M.D., Victoria, Texas.

Answer.—The glandular status of a pregnant woman is variable and differs considerably from month to month. In the first month, the chorionic gonadotropin content of the woman's tissues rises to a very high level and then decreases rather rapidly to a fairly stable level until parturition. The levels of estrogen and progesterone increase gradually from the beginning of pregnancy until term. The hormone content at any time after the first few weeks would be most difficult to duplicate by use of exogenous sources of hormones, because of the rapid destruction of these substances when they are administered by mouth or by parenteral routes. It would, perhaps, be impossible to do so without causing serious disturbances, owing to the huge quantities required to duplicate the pregnancy pattern; for example, up to 100,000 units of chorionic gonadotropin is excreted in 24 hours at one phase of pregnancy. About 10,000 units is excreted daily during the remainder of the pregnancy. One can only speculate at how many thousands of administered units would be required to duplicate this situation in a nonpregnant woman. It is possible, however, to duplicate some of the signs of pregnancy by the use of fairly small dosages of hormones; thus, administration of 5 mg. of diethylstilbestrol by mouth daily will in many cases produce a nausea that resembles that of morning sickness. Engorgement and tenderness of the breasts may be obtained with administration of 5 mg. of diethylstilbestrol with perhaps 50 mg. of progesterone daily for several weeks. The practicality of such procedures is often to be questioned.

ELECTROSHOCK NOT RECOMMENDED FOR RELIEF OF WITHDRAWAL SYMPTOMS

To the Editor:—*A recent article suggested treatment of drug addiction by immediate withdrawal of the drug plus intensive electroshock for the relief of withdrawal symptoms. Are data available on the hazards of immediate withdrawal without other treatment?*

Carroll W. Osgood, M.D., Wauwatosa, Wis.

Answer.—It is assumed that the question refers to the treatment of addiction to morphine and pharmacologically related substances. Abrupt withdrawal of morphine and related substances carries little risk to life in persons who have no serious organic disease. Abrupt withdrawal is, however, regarded as being unnecessarily cruel and deleterious to psychiatric rehabilitation of drug addicts. The recommended method of withdrawing narcotics from addicts consists of substitution of methadone for whatever drug the addict has been using, followed by reduction of the methadone dosage. This method has been described by Vogel and co-workers (*J. A. M. A.* **138**:1019 [Dec. 4] 1948). The use of electroconvulsive therapy for relieving withdrawal symptoms has not been evaluated under controlled conditions; however, since electroconvulsive therapy carries a definite, though small, risk of physical harm to the patient and since treatment of addiction by gradual reduction of narcotics carries no danger, the use of electroshock is not recommended.

EXPECTORANTS

To the Editor:—*What is the best expectorant for liquefying sputum in bronchial asthma and croup?* *M.D., Texas.*

Answer.—Because iodides concentrate in the mucous glands of the bronchi, they stimulate the latter to produce a thin secretion. Since one of the mechanisms of bronchial obstruction is the plugging of the bronchioles by tenacious sputum, the iodides frequently give relief. Potassium iodide is thus the best expectorant in asthma. Hydriodic acid, of course, also has an iodide effect. Other expectorants that may be useful in asthma or croup are those that are emetic in large doses, such as ipecac or apomorphine. The stimulating expectorants, such as ammonium chloride or terpin hydrate, are useful when the sputum is not thick, as in ordinary bronchial infection.

JOURNALS ABSTRACTED IN THE CURRENT MEDICAL LITERATURE DEPARTMENT, JANUARY-APRIL 1953

Titles have been listed or abstracts made of important articles in the following journals in the Current Medical Literature Department of THE JOURNAL during the past four months. Any of the journals, except those starred, will be lent by THE JOURNAL to individual subscribers in Continental United States and Canada and to members of the American Medical Association and its student organization. No charge is made to members, but the fee for other borrowers is 15 cents in stamps for each periodical. Three journals may be borrowed at a time for a period not exceeding five days. Requests should be addressed to the Library of the American Medical Association. Thus most of these journals are accessible to the general practitioner.

*A. M. A. American Journal of Diseases of Children. Chicago.
*A. M. A. Archives of Dermatology and Syphilology. Chicago.
*A. M. A. Archives of Industrial Hygiene and Occupational Medicine. Chicago.
*A. M. A. Archives of Internal Medicine. Chicago.
*A. M. A. Archives of Neurology and Psychiatry. Chicago.
*A. M. A. Archives of Ophthalmology. Chicago.
*A. M. A. Archives of Otolaryngology. Chicago.
*A. M. A. Archives of Pathology. Chicago.
*A. M. A. Archives of Surgery. Chicago.
Acta cardiologica. Brussels.
Acta chirurgica belgica. Brussels.
Acta chirurgica scandinavica. Stockholm.
Acta dermato-venereologica. Stockholm.
Acta medica orientalia. Jerusalem.
Acta medica scandinavica. Stockholm.
Acta neurovegetativa. Vienna.
Acta tuberculosea scandinavica. Copenhagen.
Actas dermo-sifiliográficas. Madrid.
American Heart Journal. St. Louis.
American Industrial Hygiene Association Quarterly. Chicago.
American Journal of Clinical Pathology. Baltimore.
American Journal of Digestive Diseases. Fort Wayne, Ind.
American Journal of Human Genetics. Baltimore.
American Journal of Hygiene. Baltimore.
American Journal of the Medical Sciences. Philadelphia.
American Journal of Medicine. New York.
American Journal of Obstetrics and Gynecology. St. Louis.
American Journal of Ophthalmology. Chicago.
American Journal of Orthopsychiatry. New York.
American Journal of Pathology. Ann Arbor, Mich.
American Journal of Physical Medicine. Baltimore.
American Journal of Physiology. Washington.
American Journal of Psychiatry. New York.
American Journal of Psychotherapy. New York.
American Journal of Public Health. New York.
American Journal of Roentgenol. and Radium Therapy. Springfield, Ill.
American Journal of Surgery. New York.
American Journal of Syphilis, Gonor. and Venereal Diseases. St. Louis.
American Journal of Tropical Medicine and Hygiene. Baltimore.
American Practitioner and Digest of Treatment. Philadelphia.
American Review of Tuberculosis. New York.
American Surgeon. Atlanta, Ga.
Anesthesiology. New York.
Angiologia. Barcelona.
Angiology. Baltimore.
Annales chirurgiae et gynaecologiae Fenniae. Helsinki.
Annales de médecine. Paris.
Annales medicinae internae Fenniae. Helsinki.
Annales paediatrici. Basel.
Annals of Allergy. Minneapolis.
Annals of Internal Medicine. Lancaster, Pa.
Annals of Otology, Rhinology and Laryngology. St. Louis.
Annals of the Rheumatic Diseases. London.
Annals of Surgery. Philadelphia.
Antibiotics and Chemotherapy. Washington, D. C.
Archives of Disease in Childhood. London.
Archives françaises de pédiatrie. Paris.
Archives des maladies du coeur et des vaisseaux. Paris.
Archives of Physical Medicine. Chicago.
Archivio italiano di chirurgia. Bologna.
Archivum chirurgicum neerlandicum. Arnhem.
Arizona Medicine. Phoenix.
Australasian Annals of Medicine. Sydney.
Australian Journal of Experimental Biology and Medical Science. Adelaide.
Australian and New Zealand Journal of Surgery. Sydney.
Bacteriological Reviews. Baltimore.
Beiträge zur klinischen Chirurgie. Munich.
Belgisch tijdschrift voor geneeskunde. Leuven.
Blood. New York.
Boletines y trabajos, Academia argentina de cirugia. Buenos Aires.
Brain. London.
British Heart Journal. London.
British Journal of Cancer. London.
British Journal of Dermatology. London.
British Journal of Experimental Pathology. London.
British Journal of Industrial Medicine. London.
British Journal of Ophthalmology. London.
British Journal of Radiology. London.
British Journal of Social Medicine. London.
British Journal of Surgery. Bristol.
British Journal of Tuberculosis. London.
British Journal of Urology. London.
British Journal of Venereal Diseases. London.
British Medical Journal. London.
Bulletin of the Johns Hopkins Hospital. Baltimore.
Bulletin of the Menninger Clinic. Topeka.
Bulletin of the New York Academy of Medicine. New York.
Bulletin of the World Health Organization. Geneva.
California Medicine. San Francisco.
Canadian Journal of Medical Sciences. Ottawa.
Canadian Medical Association Journal. Montreal.
Cancer. Philadelphia.
Cancer Research. Chicago.
Cardiologica. Basel.
Chirurg. Berlin.
Circulation. New York.
Clinical Science. London.
Connecticut State Medical Journal. Hartford.
Delaware State Medical Journal. Wilmington.
Deutsche medizinische Wochenschrift. Stuttgart.
Diabetes. New York.
Día médico. Buenos Aires.
Die Medizinische. Stuttgart.
Diseases of Chest. Chicago.
East African Medical Journal. Nairobi.
Edinburgh Medical Journal. Edinburgh.
Endocrinology. Springfield, Ill.
Fortschritte auf dem Gebiete der Röntgenstrahlen vereinigt mit Röntgenpraxis. Stuttgart.
Gastroenterology. Baltimore.
Gazzeta medica italiana. Milan.
Geriatrics. Minneapolis.
Glasgow Medical Journal. Glasgow.
GP (Journal of American Academy of General Practice). Kansas City, Mo.
Gynaecologia. Basel.
Hawaii Medical Journal. Honolulu.
Hiroshima Journal of Medical Sciences. Hiroshima.
Hoja tisiológica. Montevideo.
Illinois Medical Journal. Chicago.
Indian Medical Gazette. Calcutta.
Industrial Medicine and Surgery. Chicago.
International Journal of Leprosy. New Orleans.
Journal of Allergy. St. Louis.
Journal of Applied Physiology. Washington, D. C.
Journal of Arkansas Medical Society. Fort Smith.
Journal of Aviation Medicine. St. Paul.
Journal of Bacteriology. Baltimore.
Journal of Bone and Joint Surgery. Boston and London.
Journal of Clinical Endocrinology and Metabolism. Springfield, Ill.
Journal of Clinical Investigation. New York.
Journal of Clinical Nutrition. Allentown, Pa.
Journal of Clinical Pathology. London.
Journal of Endocrinology. London.
Journal of Experimental Medicine. New York.
Journal of the Florida Medical Association. Jacksonville.
Journal français de médecine et chirurgie thoraciques. Paris
Journal of Gerontology. St. Louis.
Journal of Hygiene. London.
Journal of Immunology. Baltimore.
Journal of the Indiana State Medical Association. Indianapolis.
Journal of Infectious Diseases. Chicago.
Journal of International College of Surgeons. Chicago.
Journal of Investigative Dermatology. Baltimore.
Journal of the Iowa State Medical Society. Des Moines.
Journal of the Kansas Medical Society. Topeka.
Journal of the Kentucky State Medical Association. Bowling Green.
Journal of Laboratory and Clinical Medicine. St. Louis.
Journal of Laryngology and Otology. London.
Journal of the Maine Medical Association. Portland.
Journal of the Medical Association of the State of Alabama. Montgomery.
Journal of the Medical Association of Georgia. Atlanta.
Journal of the Medical Society of New Jersey. Trenton.
Journal of Mental Science. London.
Journal of the Michigan State Medical Society. Lansing.

* Cannot be lent.

Journal of the Missouri State Medical Association. St. Louis.
Journal of the Mount Sinai Hospital. New York.
Journal of National Cancer Institute. Washington, D. C.
Journal of Nervous and Mental Disease. New York.
Journal of Neurology, Neurosurgery and Psychiatry. London.
Journal of Neuropathology and Experimental Neurology. Baltimore.
Journal of Neurophysiology. Springfield, Ill.
Journal of Neurosurgery. Springfield, Ill.
Journal of Nutrition. Philadelphia.
Journal of Obstetrics and Gynaecology of the British Empire. Manchester.
Journal of the Oklahoma State Medical Association. Oklahoma City.
Journal of Oral Surgery. Chicago.
Journal of Pathology and Bacteriology. Edinburgh.
Journal of Pediatrics. St. Louis.
Journal of Pharmacology and Experimental Therapeutics. Baltimore.
Journal of the Philippine Medical Association. Manila.
Journal of the South Carolina Medical Association. Florence.
Journal of the Student American Medical Association. Chicago.
Journal of the Tennessee State Medical Association. Nashville.
Journal of Thoracic Surgery. St. Louis.
Journal of Urology. Baltimore.
Journal-Lancet. Minneapolis.
Klinische Wochenschrift. Berlin.
Lancet. London.
Laryngoscope. St. Louis.
Lotta contro la tubercolosi. Rome.
Lyon chirurgical. Paris.
Maandschrift voor kindergeneeskunde. Leyden.
Maryland State Medical Journal. Baltimore.
Medical Annals of the District of Columbia. Washington.
Medical Journal of Australia. Sydney.
Medicina. Buenos Aires.
Medicine. Baltimore.
Mental Hygiene. Albany, N. Y.
Metabolism. New York.
Military Surgeon. Washington, D. C.
Minerva chirurgica. Turin.
Minerva ginecologica. Turin.
Minerva medica. Turin.
Minerva pediatrica. Turin.
Minnesota Medicine. St. Paul.
Missouri Medicine. St. Louis.
Monatsschrift für Kinderheilkunde. Berlin.
Monatsschrift für Unfallheilkunde und Versicherungsmedizin. Berlin.
Münchener medizinische Wochenschrift. Munich.
Nebraska State Medical Journal. Lincoln.
Nederlandsch tijdschrift voor geneeskunde. Amsterdam.
Neurology. Minneapolis.
New England Journal of Medicine. Boston.
New Jersey Medical Society Journal. Trenton.
New Orleans Medical and Surgical Journal. New Orleans.
New York State Journal of Medicine. New York.
Nordisk medicin. Stockholm.
North Carolina Medical Journal. Winston-Salem.
Northwest Medicine. Seattle.
Ohio State Medical Journal. Columbus.
Oral Surgery, Oral Medicine and Oral Pathology. St. Louis.
Pediatrics. Springfield, Ill.
Pennsylvania Medical Journal. Harrisburg.
Physiological Reviews. Baltimore.
Plastic & Reconstructive Surgery. Baltimore.
Policlinico (Surgical Section). Rome.
Postgraduate Medicine. Minneapolis.
Practitioner. London.
Praxis. Bern.
Prensa médica argentina. Buenos Aires.
Presse médicale. Paris
Proceedings of Royal Society of Medicine. London.
Proceedings of the Society for Experimental Biology and Medicine. Utica, N. Y.
Proceedings of Staff Meetings of Mayo Clinic. Rochester, Minn.
Progresso medico. Naples.
Psychiatric Quarterly. Utica, N. Y.
Psychiatry. Washington, D. C.
Psychoanalytic Quarterly. Albany, N. Y.
Psychoanalytic Review. Albany, N. Y.
Psychosomatic Medicine. New York.
Public Health Reports. Washington, D. C.
Quarterly Journal of Medicine. Oxford.
Quarterly Journal of Studies on Alcohol. New Haven, Conn.
Radiology. Syracuse, N. Y.
Review of Gastroenterology. New York.
Revista de la Asociación médica argentina. Buenos Aires.
Revista médica de Arequipa. Arequipa.
Revista médica de Chile. Santiago.
Revue de la tuberculose. Paris.
Rhode Island Medical Journal. Providence.
Rivista di clinica pediatrica. Florence.
Rocky Mountain Medical Journal. Denver.
Sang. Paris.
Scalpel. Brussels.
Scandinavian Journal of Clinical & Laboratory Investigation. Oslo.
Schweizerische medizinische Wochenschrift. Basel.
Semaine des hôpitaux de Paris. Paris.
South African Medical Journal. Cape Town.
South Dakota Journal of Medicine and Pharmacy. Sioux Falls.
Southern Medical Journal. Birmingham, Ala.
Southwestern Medicine. El Paso, Texas.
Surgery. St. Louis.
Surgery, Gynecology and Obstetrics. Chicago.
Texas Reports on Biology and Medicine. Galveston.
Texas State Journal of Medicine. Fort Worth.
Thorax. London.
Tidskrift for den norske lægeforening. Oslo.
Transactions of the Royal Society of Tropical Medicine and Hygiene. London.
Tubercle. London.
Tuberkulosearzt. Stuttgart.
Ugeskrift for læger. Copenhagen.
Ulster Medical Journal. Belfast.
United States Armed Forces Medical Journal. Washington, D. C.
Virginia Medical Monthly. Richmond.
Western Journal of Surgery, Obstetrics and Gynecology. Portland, Ore.
West Virginia Medical Journal. Charleston.
Wiener klinische Wochenschrift. Vienna.
Wiener medizinische Wochenschrift. Vienna.
Wisconsin Medical Journal. Madison.
World Medical Association Bulletin. New York.
Yale Journal of Biology and Medicine. New Haven.
Zeitschrift für die gesamte innere Medizin. Leipzig.
Zeitschrift für Kreislauforschung. Darmstadt.
Zeitschrift für Tuberkulose. Leipzig.
Zeitschrift für Urologie. Leipzig.
Zentralblatt für Chirurgie. Leipzig.
Zentralblatt für Gynäkologie. Leipzig.

SUBJECT INDEX

This is an index to all the reading matter in THE JOURNAL. In the Current Medical Literature Department only the articles which have been abstracted are indexed.

The letters used to explain in which department the matter indexed appears are as follows: "BI," Bureau of Investigation; "E," Editorial; "C," Correspondence; "ab," abstracts; the asterisk (*) indicates an original article in THE JOURNAL.

This is a subject index and one should, therefore, look for the subject word, with the following exceptions: "Book Notices," "Deaths," "Medicolegal Abstracts" and "Societies" are indexed under these titles at the end of the letters "B," "D," "M," and "S." Matter pertaining to the Association is indexed under "American Medical Association." The name of the author, in brackets, follows the subject entry.

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Am.—American
A.—Association
Coll.—College
Comm.—Commission
Comte.—Committee
Conf.—Conference
Cong.—Congress
Dist.—District
Div.—Division
Found.—Foundation
Hosp.—Hospital
Indust.—Industrial
Inst.—Institute
Internat.—International
M.—Medical
Med.—Medicine
Nat.—National
Pharm.—Pharmaceutical
Phys.—Physicians
Soc.—Society
Surg.—Surgery
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